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CONTROL OF CONCEPTION IN A SELECTED RURAL SAMPLE*

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INTRODUCTION

AS the recognition has grown that rural areas present the most urgent need for birth control, various experimental programs have been designed to carry simple methods either to women selected for certain medical indications, or to all accessible women manifesting an interest in such aid. Two assumptions dominate these projects. The first holds that the favorite methods of birth control clinics are more expensive to prescribe and to supply than available funds for rural work will allow. The second maintains that these methods are too complicated for the life routine of the rural patient and that "simpler" methods, being more acceptable, may provide a greater protection in the long run.

In 1936 a contraceptive service (1) was established under the auspices of the Mountain Maternal Health League[1] in three rural counties of Kentucky: Jackson, Madison, and Rockcastle. The objective of the project was to provide a simple and inexpensive contraceptive method to improverished mountain families within range of Berea, Kentucky, but under such conditions as would furnish an opportunity

[1] The Kentucky Maternal Health League now administers the service.

for determining the effectiveness of the service. Starting with lists drawn up by interested and supervising physicians, a public health nurse, working in the accessible areas of the three counties, instructed patients in their own homes. It was believed that clinics could not reach those in greatest need of contraceptive assistance, and that a field service would cost substantially less than a clinic service. On the view that only a simple method would be acceptable, jelly[2] alone was advised, the introduction of the jelly being accomplished by an applicator. A pelvic model was employed in instruction, and printed directions (2) were left with each patient. Payment of 10 cents per applicator and 25 cents per tube of jelly were accepted from those able to contribute to the cost of the service.

A complete reproductive history was taken at the first visit. No schedules were filled out for the unknown number who declined the service. Subsequently, each patient was visited at least every six months and follow-up entries were made in the history. In order to secure cases having had an adequate opportunity for follow-up after admission, the first 400 histories were selected for analysis. The number actually studied here is 382, eighteen histories of Negroes having been omitted in the interest of homogeneity.

A contraceptive study provides a rare opportunity for close scrutiny of the previous reproductive patterns of the particular sample. If the sample be well chosen, information of basic importance to the demographer may result. The present exploratory study offers only suggestions of the reproductive patterns in rural Kentucky, for the selection of the cases was far from random. The lack of randomness especially limits the value of the histories before enlistment, the subsequent experience being less subject to this bias. A related but secondary deficiency in the data is the absence of any basis either for an estimate of the proportion of eligible women who could be recruited into such service, or for a description of the factors associated with acceptance and rejection.

CHARACTERISTICS OF THE SAMPLE

In the history of each patient the nurse recorded color, religion, education, usual occupation of husband, whether or not the husband

[2] Lactikol B, the formula for which is: 0.05 percent oxyquinoline sulphate; 2.0 percent lactic acid; 16.0 percent glycerine; and vegetable gum base to make 100 percent by weight.

was employed on the enlistment date, and the number of months of his employment in the previous year. The present report is restricted to white women. Religion was not tabulated, but an overwhelming majority are known to have professed Protestanism. Education was recorded as completed grades, and about 50 percent of the wives had finished 8 or more years. Half of these had progressed beyond the 8th grade. At the lower end of the scale there were ten percent who had completed less than 4 years. The mean for all patients was 7.3 ± 0.15[3]. There were certain small but significant[4] differences within the sample with respect to education. Women of higher educational attainment were younger, had been married a shorter time, and were of higher socio-economic status[5]. Three status groups have been employed throughout the analysis. The first comprises 10 professional persons, 24 non-farm proprietors, managers, or officials, 19 clerks or kindred workers, and 40 skilled workers or foremen. The second includes 82 farm owners and 63 farm tenants. The third embraces 22 semi-skilled workers and 115 unskilled, mainly farm, laborers. The proportions within these status groups are roughly similar to those reported for the state in the 1930 Census (3). Approximately three fourths of the husbands were privately employed at the time of enlistment, the remainder being about equally divided between relief employment and unemployment.

The women advised ranged from 16 to 48 years of age at last birthday, with a mean age of 28.8 ± 0.36. About 8 percent were over 40 years of age at enlistment. The mean duration of marriage was 10.2 ± 0.36 completed years, and the mean number of conceptions 4.6 ± 0.16. The mean coital frequency reported at enlistment was 2.0 ± 0.06 per week, with a range of about once a month to once a day. Approximately 4 percent had never been pregnant, and 5 percent had conceived more than 10 times. The mean number of conceptions terminating in live births was 4.2 ± 0.15. For the group as a whole the mean number of pregnancies per year of marriage was 0.43 and the mean number of live births .40. In other words, the women in the sample had conceived, on the average, about once in every 28 months and a pregnancy had terminated in live birth once in every 31 months.

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[3] Standard errors are reported where pertinent.

[4] Differences are called significant here when of an order which might occur through chance less than once in 20 trials.

[5] The system of classification employed is that of Edwards (3).

Essential to an interpretation of the experience of the sample is a comparison of its fertility with that of the state and counties from which it has been drawn. Calculations based on Kentucky native white live birth schedules (4) for 1918 to 1921 and for 1929 to 1931 yield the expected rates of .30 and .26 live births per year of marriage. The observed rate of .40 reveals that the fertility of the sample is higher than that of the native white population of the state. Comparison of the three counties with the state supports the conclusion that the fertility of the sample is at least equal to, and probably greater than, the average fertility of the three counties.

REPRODUCTIVE PATTERNS BEFORE ENLISTMENT

The most important biological factors underlying the fertility of human populations are age at marriage, chance of conception, pregnancy termination, and duration of the reproductive span. The wife's age at first marriage ranged from 12 to 36 completed years, the mean being 18.2 ± 0.16. Eighty percent were married before the twenty-first birthday and 90 percent before the twenty-third. Age at marriage was higher for the better educated and for the mainly clerical and skilled workers. Pregnancy wastage not only enhances maternal mortality and morbidity but also depresses fertility. In this sample, 379 histories having 1,724 conceptions are complete for the purposes of a pregnancy termination analysis. Of these 92.4 percent terminated in live birth, 1.6 percent in stillbirth, and 6.0 percent in spontaneous abortion. The absence of induced abortion is equivocal, however, since the accuracy of such verbal reports is questionable and of the three excluded patients two may have had some voluntary pregnancy wastage.[6] Dr. Ruby H. Paine, the physician in charge of the service, has expressed the opinion[7] that induced abortion occurs among these mountain folk but reports that no estimates of its frequency have been made. Were it common

[6] Case 68 reported habitual insertion of a rubber catheter when her periods were late, and case 185 alleged that whenever her menstrual period was three days late she visited a doctor who packed the uterus. The latter claimed five abortions performed in this way. These two cases alone of the 382 admitted interference of any kind, but their histories are unsatisfactory. They might have been pregnant on none of the occasions reported by them as induced abortions, or they might have been pregnant each time. Even in the latter event no estimate could be made for case 68.

[7] In a private communication.

among these women, some induced abortions would probably have been falsely reported as spontaneous. This seems unlikely, however, since the percentage of spontaneous abortions is in fact well *below* the careful estimates of 9 to 10 percent made by Wiehl (5) on the basis of urban samples.

In the exploration of the effect of contraception and other factors upon the chance of conception, the basic concept is that of exposure to the risk of conception. As developed by Pearl (6), exposure to the risk of conception is the time, expressed in months or years, which a woman spends in the conjugal state and during which conception is physiologically possible. A division is thus made between some and no possibility of conception, and each month of married life appropriately classified. This twofold division makes no provision for the large differences in the actual likelihood of conception among months given equal weight as months of exposure. For example, months of postpartum amenorrhea and lactation are usually included as exposure-months, although the risk of conception is very low at this time. Circumstances which preclude conception are pregnancy, abstinence, sterilization, and demonstrated sterility. The principal deduction is for pregnancy. Parallel to the classification of the months of exposure is the enumeration of conceptions, each month of exposure having either one or none.

Statistical measures for summarizing the exposure and pregnancy experience of a group are several, but that employed here is a weighted mean rate calculated directly from the ratio of the sum of the conceptions to the sum of the exposure for any category. For convenience this ratio is multiplied by 1,200, making it a pregnancy rate per 100 woman-years of exposure to the risk of pregnancy $\left(\frac{1{,}200 \times \text{pregnancies}}{\text{woman-months of exposure}}\right)$. In the present instance, for example, the total experience before admission yielded 30,180 months of exposure and 1,662 conceptions for which exposure could be calculated. This may be expressed as a pregnancy rate of 66 pregnancies per 100 woman-years of exposure. All tables give exposure in months and rates as pregnancies per 100 years of exposure. Comparison with three published series (7, 8, 9) indicates that the fertility of the present sample lies within the range of expectation.

Pregnancy rates for the three broad socio-economic status groups yield but one reliable difference,[8] the lower rate of 60 for the skilled and clerical workers taken in contrast to the rates of 68 and 70 for the two farming groups. The difference between educational groups is

TABLE 1

Risk of conception before admission, by contraceptive practice, order of pregnancy, and duration of marriage

ORDER OF PREGNANCY AND DURATION OF MARRIAGE	CONTRACEPTIVE PRACTICE				
	No contraception practiced				Some † contraception practiced
	Number of women	Months of exposure	Number of pregnancies	Rate*	
All pregnancies.....	352	23,035	1,500	*78*	*27*
First pregnancy.....	314	2,606	305	*140*	**
Subsequent pregnancies, by years of marriage					
Total.....	318	20,429	1,195	*70*	*26*
0–4	300	7,181	483	*81*	*24*
5-9	221	6,421	374	*70*	*24*
10-14	141	4,190	198	*57*	*28*
15-19	71	1,864	100	*64*	*29*
20-29	32	773	40	*62*	**

* Pregnancies per 100 woman-years of exposure.

$$\text{Rate} = 1{,}200 \times \frac{\text{Conceptions}}{\text{Exposure-months}}.$$

† Exposure and pregnancies appear in Table 3, columns 3 and 4.

** Less than 500 months of exposure upon which to base a rate.

also significant, the pregnancy rate being 74 for women with less than seven, and 59 for women with seven, or more, completed grades. Very large differences are found when cognizance is taken of contraceptive practice. In Table I pregnancy rates for each duration of marriage

[8] The method of the contingency table, employing critical values for χ^2, has been used here. For fourfold tables Yates' correction and the Fisher and Yates (10) approximation of the exact treatment of fourfold tables have been employed when appropriate.

interval are shown by contraceptive experience. The chance of conception while contraception was attempted was 27 pregnancies per 100 woman-years of exposure, or approximately a third of the rate of 78 when no contraceptive effort was made. The difference represents a reduction of about 65 percent in the chance of conception *before* advice was given. ***This finding makes it quite clear that the group already had the means for controlling its reproduction without having made extensive use of it.*** However, it must also be observed that the indicated protection is unexpectedly high in comparison with previous reports.

Perhaps the best measure of the relative extent of contraceptive practice is the proportion of exposure during which contraceptive precautions were taken. This index shows that one fourth of the exposure prior to enlistment was contraceptive, the proportion rising with increasing duration of marriage. Before the first pregnancy 12 percent of the exposure was contraceptive, while 37 percent of the exposure of those married for 20 or more years was so classified. Measures correlating well with standards of living, such as age at marriage, education, and socio-economic status show clearly the tendency of the more favored patients to report higher percentages of contraceptive practice. Thus the percentages are 21 for those married before nineteen, and 29 for those married thereafter; 14 for women with less than seven completed years of schooling, 28 for women with seven to nine years, and 48 for women with ten or more years; and 13 for semi- and unskilled workers, 23 for farm owners and tenants, and 41 for skilled and clerical workers.

Not all contraceptive practice is of equal significance for the reduction of pregnancy risk. While 61 percent denied ever having practiced contraception, 16 percent had at some time employed the condom, 12 percent withdrawal, 12 percent douche, 3 percent jelly, 2 percent some variety of pessary, 2 percent miscellaneous methods, 1 percent pessary and jelly, 1 percent suppository, and less than half of one percent the "safe period." Withdrawal, douche, or condom were reported by 33 percent of the patients, or 85 percent of the contraceptors.

Although the contraceptive practice of the group resulted in an average reduction in the chance of conception of about 65 percent, not all methods were used with the same success. Stated in terms of reduc-

tion in the chance of conception when contraception was not used,[9] the estimated protection with each was: condom 80 percent; other and combination methods 71 percent; withdrawal 60 percent; and douche 46 percent. Comparison of the socio-economic groups shows that the mainly skilled group obtained significantly more protection from its contraceptive endeavor than did the farming groups. The effectiveness percentages are roughly 70 and 60, and the increment is associated with a greater dependence upon the condom by the mainly skilled group.

Resort to contraception depends upon many factors. It has been shown by Stix (11) that the risk of conception women experience without attempting to prevent conception may be a factor in this decision. She found that contraceptors had encountered a greater risk of conception before taking up contraception than had those who remained noncontraceptors. This hypothesis has been tested on the present series since, if generally valid, it throws important light upon the selective process impelling some women to practice contraception. Division of the noncontraceptive exposure into two parts, that of the women who never practiced contraception, and that of women with at least some contraceptive experience, results in large and consistent differences for the first pregnancy and for each duration of marriage interval; the total rates are 72 and 100. This result is confirmed by a second analysis in which the noncontraceptive experience within each duration of marriage interval was studied in relation to whether or not contraception was practiced in the following interval. From these two tests it is evident that those who took up contraception had a basic noncontraceptive rate 30 to 40 percent higher than those who failed to do so. Were the comparison extended beyond the present sample to women who *refused the advice* offered but who had never practiced contraception, it seems likely that even lower noncontraceptive rates might be obtained (12).

ACCEPTANCE OF THE SERVICE

In the absence of immunological or other long-term protection, conception is avoided only by the skilful and assiduous application of a contraceptive routine. This circumstance underlies the paramount

[9] The rate employed is that for the whole sample. The efficiency would be somewhat higher if based on the frequency of conception of the contraceptors alone before they took up contraception. The pregnancy rates for each method are given in column 5 of Table 4.

importance of those many factors which are currently incorporated in the notion of acceptability. Investigation has demonstrated that the difference between any and no method, or between skilful and unskilful practice of a method, overshadows differences among methods and even produces variation in protection from a single method.

Twenty-seven patients, or about 7 percent of the total number advised, could not be located after the first visit. The average duration of follow-up was 16 months for the entire sample of 382 patients, and 18 months for the 355 with some follow-up. Eighty percent of all patients were followed up for a year or more, and 20 percent for two years or more, the distribution of follow-up being largely determined by the fact that enlistment covered a period of two and a half years, and by the fact that no particular effort was made to keep in touch with patients who expressed their dissatisfaction with the service. A definite bias is introduced by the relationship between acceptability and duration of follow-up, one for which some correction is imperative.

Acceptability can be measured (13) only in terms of its component elements. Employed here as indices are:

(1) proportion ever giving the method a trial;
(2) proportion of patients alleging regular use of the method;
(3) proportion of exposure after enlistment during which the method was employed alone as a contraceptive; and
(4) proportion of users at successive intervals following enlistment.

A review of the 355 cases with follow-up shows that 62, or 17 percent, never gave the method a single trial, and that an additional 7 probably, although not certainly, never used the method. Various factors examined were found to be unrelated to lack of trial: socio-economic status, age of wife, education of wife, and type of contraceptive method used prior to enlistment by contraceptors. However, the contraceptive history *before* admission showed a significant relationship. Twenty-five percent of the prior noncontraceptors, and 11 percent of the contraceptors, failed to try the jelly furnished. Among the contraceptors education, but neither age nor socio-economic status, showed a significant and positive relationship to trial.

A second approach to the relative acceptability of the prescription places emphasis upon the mode of use. When the patients who used the jelly are separated into those who reported infallibly regular use

and all others, 54 percent of the cases fall into the category of regular users. When the proportions are compared for prior contraceptors and noncontraceptors, no difference is found. This is also characteristic of socio-economic status, education, age of wife, months jelly was employed, and coital frequency at enlistment. In short, none of these more important variables appears to be associated with regularity in these observations. This finding does not constitute proof of the absence of such a relationship, if for no reason other than the uncertain value of the verbal report on regularity. It was found that those women who reported irregular use of the prescribed method had experienced a high pregnancy risk *before* admsision while practicing contraception. The prior rate was 23 for subsequently regular users, and 34 for irregular users.

The third measure of relative acceptability is the proportion of time spent practicing the prescribed method. It is preferable, however, first to compare the various groups with respect to the degree to which they made any attempt to control conception. Prior contraceptors sought protection during 92 percent of their exposure, and prior noncontraceptors 75 percent. Among the contraceptors there was a clear educational differentiation not found among the noncontraceptors, those with the poorest education reporting a reliably less extensive use of contraception after admission. A significant relationship was also found for socio-economic status when no subdivision by prior contraceptive use was made. The mainly skilled and white-collar group reported 88 percent of the exposure following enlistment as contraceptive, the farm owners and tenants 83 percent, and the unskilled 78 percent.

The best measure of the relative dependence upon the prescribed method is the proportion of exposure with jelly alone, not in the total, but in the exposure during which protection was sought. In this way the preceding differences in resort to contraception are controlled. The percentage of jelly alone exposure to total contraceptive exposure is 67 for the entire sample. A striking and reliable difference exists between prior contraceptors and noncontraceptors, indicating that patients with previous contraceptive experience were less satisfied with the contraceptive merits of jelly alone than were the prior noncontraceptors. Significant differences among educational groups exist for both contraceptors and noncontraceptors, women with more education reporting less extensive use of the jelly alone method. A similar result ensues when the indices for the three socio-economic status groups are

compared without separating the contraceptors from the noncontraceptors.

In most series follow-up ends when the patient stops using the prescribed method. In consequence, the experience of women followed up a given length of time after enlistment may fail to reflect the actual likelihood of use at this time. However, by the life table principle, a

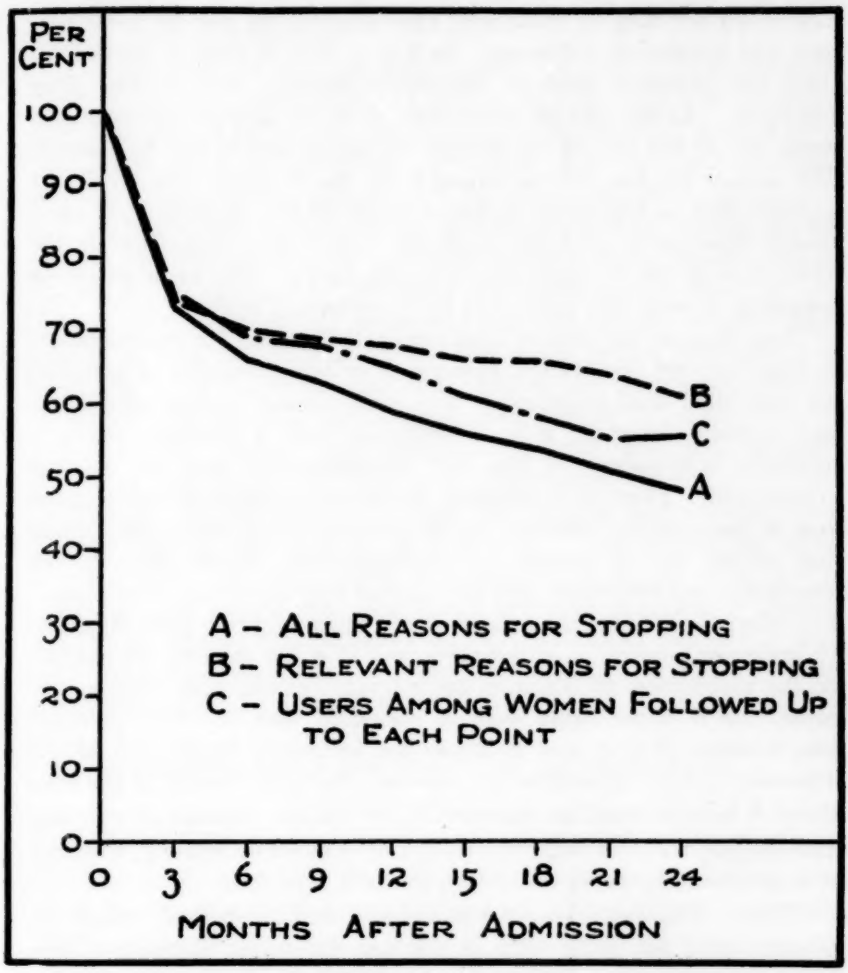

FIG. 1. PERCENTAGE OF PATIENTS USING METHOD AT INTERVALS AFTER ADMISSION

table of users may be constructed which is independent of variation in duration of follow-up provided that the risk of stopping in each month is known. From a cumulative distribution giving the number using at or beyond any month, and the number dropping out in that month, the risk of stopping has been computed for each month. This schedule of risk may be applied to the sample group as the only factor operating to diminish it, whereas actually the users in any given month are diminished not only by those who have stopped but also by those who have had insufficient follow-up. In Fig. 1, line A gives in percentage form the estimated users at successive intervals of 3 months after admission. At the end of 12 months about 60 percent are estimated users, but at the end of 24 months the percentage is but 50 percent. The large initial loss reflects refusal to try the method. The possibility and direction of bias from estimates based on the proportion of users among those with a given length of follow-up is indicated by the divergence of line C from line A in the figure. The error might be greater in a series less influenced by experimental interest.

Some reasons for non-use also terminate the need for contraception. Among these are sterilization, menopause, or other cessation of exposure not associated with pregnancy. For comparisions among populations and methods, however, it is desirable to have a measure which is relatively independent of the risk of conception with the method investigated. There is a residual group of patients whose non-use may be described as "relevant" to the acceptability of the method, while that arising out of absence of exposure, lack of supplies, desired pregnancy, and pregnancy with the method may be termed "irrelevant." For other comparisons the criteria of relevance might be quite different. If irrelevant non-use is disregarded, and if a life table is constructed on the assumption of cessation for relevant reasons only, then a new curve, line B in the figure, may be compared with A. The widening area between lines A and B shows the increasing importance of the irrelevant factors as reasons for non-use. In other words, if the area above A be considered as representing the relative amount of non-use, then the proportion falling between A and B increases steadily, although at no point within the 24 month range does it reach half.

Of the 286 patients known ever to have tried the method, 108, or 38 percent, were not using jelly at the last follow-up, although a few indicated that they would resume the method in the future. Of the patients who were not using the method at the last visit 27 percent

associated non-use at least partly with preference for other methods or with lack of confidence not bred of undesired conception, 11 percent with messiness, 3 percent with fear of injury, and 2 percent with burning. In the entire sample of 286 users the messiness complaint was made by 6 percent, and burning by 2 percent. If those with conceptions during apparent use be added to the patients expressing distrust or preference for other methods, then it may be said that about 15 percent of the 286 users, or 40 percent of those who stopped, gave these reasons for discontinuance.

The foregoing evidence on acceptance of the service bears summary. With an initial refusal rate of 17 percent, it is perhaps not surprising that an estimated 50 percent were not using the prescribed method after two years. Prior contraceptors, and among them the better educated, were more willing than others to try the method advised. No descriptive characteristics appeared to be related to regularity of use of the method, but the regular users did have a more successful contraceptive history *before* enlistment. Although contraceptors and those favored with respect to educational attainment accepted the new method more readily at first, and throughout the subsequent period practiced more contraception of all kinds, they relied less on the jelly method during their contraceptive practice, shifting more readily to other methods. Socio-economic status, while unrelated to trial, was also inversely associated with use of the jelly as a contraceptive, for the skilled and white-collar families used it least and the unskilled farm families used it most during their contraceptive practice. The conclusion seems inescapable that the more critical and experienced women of the group, while eager to try the new method and much more willing to practice contraception, were more prone to abandon it for other methods of their own choosing, and that the less experienced and less favored were more dependent upon the method given them.

CONTRACEPTION AND THE CHANCE OF CONCEPTION AFTER ADMISSION

Over the follow-up period, which averaged 18 months in length, the chance of conception declined from the previous pregnancy rate of 59 for second and subsequent pregnancies to 26, a drop of 56 percent. Table 2 gives the two sets of rates by duration of marriage. It will be seen that reductions occurred in each interval, and that they are of the same order. Not all patients achieved the same reductions, however.

TABLE 2

Comparison of risk of conception before and after admission, by order of pregnancy, and by duration of marriage, without regard to contraceptive practice

ORDER OF PREGNANCY AND DURATION OF MARRIAGE	BEFORE ADMISSION				AFTER ADMISSION			
	Number of women	Months of exposure	Number of pregnancies	Rate*	Number of women	Months of exposure	Number of pregnancies	Rate*
All pregnancies....	373	30,180	1,662	*66*	319	4,619	101	*26*
First pregnancy....	330	2,957	322	*131*	12	146	4	**
Subsequent pregnancies, by years of marriage								
Total.....	355	27,223	1,340	*59*	312	4,473	97	*26*
0-4.....	343	9,005	519	*69*	73	747	20	*32*
5-9.....	265	8,895	424	*57*	93	1,176	25	*26*
10-14.....	173	5,478	228	*50*	107	1,324	29	*26*
15-19.....	84	2,618	118	*54*	50	627	14	*27*
20-29.....	42	1,227	51	*50*	47	599	9	*18*

* Pregnancies per 100 woman-years of exposure. Rate $= 1{,}200 \times \frac{\text{Conceptions}}{\text{Exposure-months}}$.

** Less than 500 months of exposure; rate not computed.

The socio-economic groups differ in the expected direction. The mainly skilled group reduced its risk of conception about 75 percent, the farm owners and tenants about 60 percent, and the unskilled about 50 percent. Similarly, those who had never practiced contraception before enlistment registered a reduction of about 50 percent, while those with prior contraceptive experience reduced their risk about 60 percent.

The decline in the frequency of conception may be analyzed at different levels. The actual practice of contraception increased greatly, the percentage of contraceptive exposure rising from 25 to 83. Since present contraceptive methods require the conscious and sedulous participation of the patient, and since the patients were selected for interest in family limitation, it might be argued that this result could

be expected from selection alone. This view is untenable. Observations (12, 14) in rural areas elsewhere in the Southern Appalachians have shown that 30 to 50 percent of all white married women of reproductive age may be expected to accept such a contraceptive service. If such a large proportion of women, and a proportion, moreover, of probably greater than average fertility, is at any time ready to shift voluntarily to contraception, and would in fact do so without the benefit of a contraceptive service, the present high fertility of the Southern Appalachian region would have disappeared within the past decade.

TABLE 3

Risk of conception while practicing contraception before and after admission, by order of pregnancy and by duration of marriage

ORDER OF PREGNANCY AND DURATION OF MARRIAGE	BEFORE ADMISSION				AFTER ADMISSION			
	Number of women	Months of exposure	Number of pregnancies	Rate*	Number of women	Months of exposure	Number of pregnancies	Rate*
1	2	3	4	5	6	7	8	9
All pregnancies....	144	7,145	162	*27*	288	3,826	54	*17*
First pregnancy....	28	351	17	**	11	126	1	**
Subsequent pregnancies, by years of marriage								
Total.....	135	6,794	145	*26*	276	3,700	53	*17*
0-4.....	81	1,824	36	*24*	64	602	11	*22*
5-9.....	89	2,474	50	*24*	93	1,009	14	*17*
10-14.....	45	1,288	30	*28*	87	1,094	14	*15*
15-19.....	26	754	18	*29*	49	507	8	*19*
20-29.....	12	454	11	**	41	488	6	**

* Pregnancies per 100 woman-years of exposure. Rate $= 1{,}200 \times \frac{\text{Conceptions}}{\text{Exposure-months}}$.

** Less than 500 months of exposure; rate not computed.

Before enlistment the contraceptors in the series had demonstrated an ability to lower their frequency of conception to 26 pregnancies per 100 woman-years of exposure while practicing methods of their own selection. The comparable rate after admission is 17, about 35 percent lower. The rates are given in Table 3, specific by duration of marriage, and show differences for all intervals. Taken together the differences are statistically significant by the criteria employed here.

Not all groups within the sample, however, significantly increased their protection while contraception was practiced. Among the socio-economic groups, only the farm owners and tenants made a significant gain. For the three broad educational groups the contraceptive risk after admission was in each case lower than the corresponding risk before admission, but only that for the lowest educational class, a drop from 42 to 20, is not in accordance with chance expectation. When a comparison is made between those who ever and those who never practiced contraception *before* enlistment, the pregnancy rates of 13 and 21 *after* enlistment are found to be reliably different for the amount of experience involved, about 150 and 160 woman-years of exposure respectively. Other characteristics might show differences not revealed by the above analysis, but the foregoing should be sufficient to show, first, that the gain in protection, while considerable in relative amount, was somewhat less than might have been anticipated, and second, that while the most favored groups continued to have the greatest security, the patients with the highest pregnancy rates before admission derived large relative and absolute gains.

Since two thirds of the contraceptive effort after enlistment depended upon the jelly alone method, it might be expected that high protection was realized during its use, but that resort to other methods had lowered the average risk after admission. As Table 4 shows, however, the risk with jelly alone was actually higher, although insignificantly so, than with all other methods. When the pregnancy rate with jelly alone is tested against the chance of conception for second and subsequent pregnancies during contraceptive practice *before* enlistment, the difference is found to be of a magnitude which might easily arise through random variation. Although the protection with all methods was definitely superior in the later period, no single method shows a significant improvement such as a larger body of observations would be expected to show. In general, the findings are in accordance with the view that a contraceptive method was provided by means of which

the patients secured a protection no higher than they had obtained with methods of their own choosing. The contribution of the service, therefore, consisted in the encouragement to practice contraception.

The experience of the sample with jelly alone was further studied for rate differentials within the group. The factors studied were socio-economic status, education, prior contraceptive history, and

TABLE 4

Risk of conception before and after admission, by contraceptive practice, including method employed, for second and subsequent pregnancies only

CONTRACEPTIVE PRACTICE	BEFORE ADMISSION				AFTER ADMISSION			
	Number of women	Months of exposure	Number of pregnancies	Rate*	Number of women	Months of exposure	Number of pregnancies	Rate*
1	2	3	4	5	6	7	8	9
Total experience...	355	27,223	1,340	*59*	312	4,473	98	*26*
No contraception employed	318	20,429	1,195	*70*	103	773	45	*70*
Contraception employed								
All methods.....	135	6,794	145	*26*	276	3,700	53	*17*
Withdrawal	36	1,862	43	*28*	27	246	3	**
Douche	40	1,822	58	*38*	7	68	0	**
Condom	51	1,795	21	*14*	42	336	2	**
Jelly	3	24	1	**	220	2,451	41	*20*
Other	23	1,291	22	*20*	60	599	7	*14*

* Pregnancies per 100 woman-years of exposure. Rate $= 1{,}200 \times \frac{\text{Conceptions}}{\text{Exposure-months}}$.

** Less than 500 months of exposure; rate not computed.

alleged regularity of jelly use. Although all these elements yield small differences in the expected directions, only the last, regularity, produces a difference outside the range of chance expectation. For this test the sample was divided into two parts, those jelly alone users who claimed perfect regularity, and those who admitted one or more

omissions. The former had a pregnancy rate of 14, and the latter one of 33. Corresponding to this finding is the report of more than half the 41 patients with jelly alone conceptions that pregnancy may have been associated with omissions. The question about regularity was an integral part of the follow-up schedule for each visit and was asked of each patient regardless of her pregnancy experience. This would seem to be an elementary requirement in the collection of such data, for the classification of conceptions on the basis of regularity allows for no estimate of the amount of risk in each group. If accurate estimates are to be made of the influence of regularity, obviously a superior basis of classification is essential, for the category used here for contrast with regular use is itself rather heterogeneous with respect to the regularity of jelly use for each month of exposure. Until more confidence can be placed in the basic observations, however, the value of more refined treatment is questionable. From the standpoint of scientific accuracy in the recorded schedules it is unfortunate that the information is obtainable only in a social and psychological context which may prejudice the response of the patient. Any bias is doubtless in the direction of reducing the spread between the two risks. It is for these reasons (15) that no more emphasis has been placed on this factor in the present report, despite its obviously focal importance in determining the risk of conception.

SUMMARY

The reproductive histories of 382 selected rural and mainly farm wives have been analyzed for their bearing on the voluntary control of conception both before and after the institution of a contraceptive service.

Before enlistment

(1) The total pregnancy rate was 66 pregnancies per 100 years of exposure. Thirty-nine percent of the women reported some contraceptive practice, and 25 percent of all the exposure was contraceptive.

(2) The uncontrolled chance of conception, after the first pregnancy, was 70, which is below that reported for some urban clinic series. Before taking up contraception the contraceptors had experienced a risk 30 to 40 percent above that of women without any contraceptive experience.

(3) During contraceptive practice the pregnancy rate was 26, an efficiency of 63 percent, and the calculated effectiveness of the condom was 80 percent, withdrawal 60 percent, and douche 46 percent.

After enlistment

(1) Seventeen percent refused to try the method advised.

(2) At the last follow-up 62 percent of those who had tried the method were continuing its use. It was estimated that 60 percent of the women advised were using the method after 12 months, and 50 percent after 24 months.

(3) The percentage of exposure classified as contraceptive increased from 25 to 83 in the follow-up period, which averaged 18 months, and the chance of conception declined 56 percent.

(4) While contraception was practiced in the follow-up period, frequency of conception fell 35 percent below the contraceptive rate *before* admission. Although the lowest pregnancy rates continued to be those of patients with high previous protection, large gains were made by women whose prior contraceptive practice had been less efficient.

(5) The prescribed method accounted for 55 percent of all the exposure after admission, and for 67 percent of the contraceptive exposure.

(6) While the prescribed method was employed the pregnancy rate was 20, insignificantly different from the risk experienced with all other methods either before or after enlistment. The only factor reliably associated with differential risk while using the prescribed method was stated regularity. The pregnancy rate while the prescribed method was used was 14 for the 54 percent who alleged regular use in comparison with 33 for all other patients.

(7) In general the interpretation placed upon the results is that the patients secured no higher rate of protection with the jelly method than with methods of their own choosing, but that the service made a large contribution to the control of conception in encouraging their practice of contraception.

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SOMATOLOGY OF THE SCHIZOPHRENIC PATIENT

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INTRODUCTION

THE primary purpose of this study is to investigate the somatological status of individuals who are, clinically, characterized by the schizophrenic reaction. A second purpose is to determine whether somatological status is in any way a significantly associated factor in the genesis and clinical course of the schizophrenic process. The investigation is based on 193 patients, the total resident population of schizophrenic women in the Spring Grove State Hospital in Catonsville, Maryland, at the time the study was begun (1937-38), and on a control group of thirty-two non-psychotic women, members of the nursing staff of the Spring Grove State Hospital. The basic problems of the inquiry may be stated as follows: What, if any, are the significant differences in somatic structure between the schizophrenic and non-schizophrenic individuals in this material? What, if any, are the differences in psychiatric status between patients of one type of somatic organization and those of another? The first question is essentially descriptive in its connotation, but as such leads to a more precise knowledge of the biological framework constituting the schizophrenic personality. The second question penetrates more deeply and is intimately concerned with pathogenesis. A study of the structure and functioning of single systems in looking for etiologic and pathogenic factors in the schizophrenic reaction has yielded little basic information. It would seem that the integrated expression of these "part" functions, that together make up the body *organization* or somatic habitus which *constitutes* the individual, should not be ignored.

The term *constitution* has been used in various senses in the literature, at times with reference strictly to type of personality reaction, at times as synonymous with body structure or habitus. It seems important at

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the outset to consider the meaning of constitution as it will be here used. The modern concept of constitution has been defined by Pearl (1933, p. 46) as follows: "The biological constitution (total personality) of an individual at a given moment depends directly and immediately upon and is determined by, at least, A — The present functional condition (tonus and balance) and state of activity of the endocrine system, the autonomic nervous system, the vascular and vasomotor system, the central nervous system (psychic influence). All of which in turn depend in part upon B, the physical chemistry of the blood, and the innate degree of perfection (biological worth) of the anatomical structure in general, and of each particular organ, and the age of the individual." All derive from "the inherited kind and quality of biological organization and from the environmental circumstances and forces, past and present." It is interesting to note that this point of view which regards the biological constitution as, in fact, the total personality, is essentially identical with that of Meyer who regards the functioning personality as an integration of all of the "part" systems of the body. The study of an individual in terms of constitution, more concretely in terms of somatic habitus, is therefore a direct and integral part of the study of his functioning personality. This point, indeed, is emphasized by Pearl and Ciocco (1934, p. 650) when they define the objectives of the study of human constitution as: " . . . the discovery of stable, organic correlations (integrated biological relations) between the morphological, physiological, psychological and pathological characteristics of the individual, and eventually the precise numerical measurements of such correlations."

The term *habitus* is used in the sense of bodily configuration, somatic structure, or somatic organization, and refers to one aspect of an individual's constitution, just as personality refers to another. Both are aspects of the same thing (the individual constitution); are related as an integral whole; and have only academic meaning apart from each other. Various writers on human constitution, in classifying habitus into different types, have introduced their own nomenclature. Wertheimer and Hesketh (1926, pp. 10-11) have tabulated these various names as they correspond to each other and they will not be discussed here. In the present study, variations in human habitus are recognized as distributed in a continuous manner (Pearl and Ciocco, 1934) with, at one extreme, a relative preponderance of vertical over horizontal measurements (asthenics), at the other extreme a relative preponderance of horizontal over vertical measurements (pyknics), and with inter-

mediate relationships between vertical and horizontal dimensions lying between the two extremes. An arbitrarily made triple division of this continuous distribution leads to the usual classification of body habitus: asthenic, intermediate, and pyknic. A fourth group, the dysplastic, comprises individuals of uneven somatic development whose bodily conformation presents a mixture of two or more of the other types. While this terminology follows Kretschmer closely, the idea of continuity in variation of somatic habitus emphasized here, has perhaps been less widely appreciated.

EARLY EFFORTS TO CORRELATE BIOLOGICAL CONSTITUTION WITH MENTAL DISEASE

Esquirol in 1816 and Morel in 1857 had each pointed out an association between the external appearance of their patients and the character of their illness. However, that psyche and soma seemed to go hand in hand had been recognized since the time of Hippocrates. An excellent, concise account of the historical background of the modern study of constitution is given by Ciocco (1936) to whose paper the reader is referred. He notes that, among those writers who pointed out a relationship between mind and physiognomy were Aristotle, Polemonis, a Greek of the third century A.D., Adamantius, a Jewish physician of the fourth century A.D., Giambattista della Porta, a Neopolitan philosopher of the sixteenth century, and J. G. Lavater, a Swiss Protestant minister of the eighteenth century. Not too distantly related were the phrenologists F. J. Gall (1757-1828) and J. C. Spurzheim (1776-1832) whose work ultimately became the stock-in-trade of quacks. Attempts to relate body structure and disease date back to Hippocrates and Galen and their classification of human beings into four types on the basis of temperament: phlegmatic, plethoric, bilious, and melancholic.

Although it has long been recognized that each individual in his somatic structure, in contrast to his capacity as a functioning personality to project himself to formidable dimensions, is strictly contained within definite and relatively fixed anatomical limits, the only attempts to ascertain these limits in quantitative terms until modern times were, as Ciocco points out, made by painters and sculptors. The modern study of constitution on a scientific basis dates from the work of Beneke (1878), and is subsequently associated with the work of De Giovanni,

Viola, and Pende. More recently, in this country the contributions of Draper and of Pearl and Ciocco have been outstanding.

Kretschmer's book, published in Germany in 1921, was the first attempt to demonstrate a relationship between somatic habitus and the schizophrenic psychosis, and one of the first attempts to associate physical habitus and mental disease. In the field of psychiatry it is the outstanding and perhaps still most important single contribution to the problem of constitution and psychiatric disease. Largely by clinical impression Kretschmer noted and pointed out that the asthenic type of body build particularly, and to a lesser extent the athletic type, seemed to be associated with a schizoid pre-psychotic personality and the schizophrenic psychosis, whereas the pyknic type of body build was found associated with a cycloid pre-psychotic personality and the manic-depressive psychosis. Kretschmer's methods, both in determining his data and in presenting them — points discussed in more detail subsequently — are open to criticism (Burchard, 1936), but his contentions proved tremendously stimulating, and the greatest importance of his work perhaps lies in the strong impetus he gave to further research.

In the decade following Kretschmer's book, a vast amount of work was done on the problem of somatic habitus and psychiatric disease, particularly in Germany. This was in large part patterned after Kretschmer, with a view to proving or disproving his conclusions. Among those whose results agreed essentially with Kretschmer may be mentioned the Germans Sioli and Meyer (1922), Oliver (1932), Jakob and Moser (1922), Michei and Weeber (1924), Henckel (1924), Weissenfeld (1925), von Rohden (1926), Wyrsch (1924), and van der Horst (1924); the Italian Verciani (1922); the Russians Schubert, Makarow, Andrejew, and Guber-Gritz; the Poles Matecki and Szpidbaum (1927); and the Americans Shaw (1925), Wertheimer and Hesketh (1926), Farr (1928), and Adler and Mohr (1928). Among those writers whose results made them critical of Kretschmer may be mentioned the Germans Kolle (1925), Möllenhoff (1924) and Gruhle (1926), and the American Wells (1938). Corroboration in part of Kretschmer's tenets is reported by the Americans Raphael, Ferguson and Searle (1925), and Burchard (1936). Pertinent data from these investigators will be considered at appropriate points in subsequent sections. It may be stated that, notwithstanding the attention it has received, the subject of psycho-somatic relationships in schizophrenia

is still controversial and suffering from a surplus of inadequate data, the result in large part of inadequate procedures of investigation and inadequate grasp of the objectives sought.

With regard to the general problem of somatic habitus and psychiatric disease, Lewis (1936, p. 133) writes: "The widely quoted work of Kretschmer on the correlations of bodily configuration is a step in the right direction, although it has received some criticism, directed principally toward the method used, and with the exception of the studies of Wertheimer and Hesketh and of Raphael has not been applied in a practical sense to any notable degree in this country." He continues (p. 135): "More research is indicated on the kretschmerian concepts of physical build and personality correlations, as evidenced by the present state of divided opinions among the scientific authorities as to the value and reliability of the studies made in the past."

THE MATERIAL

The general characteristics of the 193 schizophrenic women comprising the present sample are described below.

It was found that 80.7 percent are American-born. The remaining 19.3 percent includes ten different nationalities (Russian, Italian, German, Lithuanian, Polish, Austrian, English, Czechoslovakian, Galician and Roumanian). In comparison with the figures for the population of Maryland which represent the native-born as 93.0 percent of the total white population and the foreign-born as only 7.0 percent, the proportion of foreign-born in the present sample, by statistical test, is significantly higher. (A difference which is "statistically significant" is one which would be unlikely to occur merely through the operation of chance. Technically, a difference is termed significant if it is three or more times its probable error.) In view of the strain involved in adaptation to the mores and language of a new country, this difference does not seem remarkable. It would not seem to be a factor of importance in the present somatological study.

The patients in this group are all adults, ranging in age from fifteen to sixty-nine: 42 percent are forty years old or under; 58.0 percent are forty-one or over. The mean age is 43.5 years.

In connection with the age factor, data on the age of onset of the present illness and the length of time since onset of the present illness may be briefly mentioned. In the present sample no patients developed their illness before the age of fifteen, nor after the age of sixty. The

mean age of onset is 30.3 years. The length of time since onset of the present illness in these patients ranges from a few weeks to over thirty years. The mean interval since onset is 13.2 years.

The age range of the control group is from eighteen to forty-two, with a mean age of 26.3 years. That this is a younger group than the schizophrenic is to be kept in mind.

METHODS USED IN SOMATOLOGICAL STUDY

The following anthropometric data were obtained on each of the 193 schizophrenic women and on the thirty-two non-psychotic women comprising the control group, and recorded on the standard form shown below: three head measurements (girth, breadth and length); four vertical measurements of the body (sitting height, stature, sternum length, and xiphoid-symphysis length); three horizontal body measure-

CONSTITUTIONAL STUDY OF PSYCHOSES

Spring Grove State Hospital

Catonsville, Maryland

SOMATIC DESCRIPTION

Name_____ Case No._____
(Last) (First)

Anthropometric:

Head girth_____cm.
Head breadth_____cm.
Head length_____cm.
Sitting height_____cm.
Stature _____cm.
Biacromial dia._____cm.
Bicristal dia._____cm.
Xiphoid-symphysis _____cm.
Sternum length_____cm.
Wrist width, right_____cm.
left_____cm.
Chest depth_____cm.
Chest girth_____cm.
Umbilical girth_____cm.
Weight_____lbs._____kg.

Pulse (at end of examination)_____

Trunk
0 1 2 3 4 5 6 7 8 9 10 11 12

Legs
0 1 2 3 4 5 6 7 8 9 10 11 12

Habitus: asthenic, intermediate, pyknic, dysplastic

Hand grip: Rt._____kg.
Lt._____kg.

Date of examination_____
Time of examination_____
Examiner _____

ments (biacromial width, bicristal width, and chest depth); two circumferences (chest girth and umbilical girth); wrist width and weight. These particular measurements were selected out of many possibilities as the most fundamental, simple and accurately obtainable. In addition to these anatomical variables, hand grip was determined as a possible measure of psychomotor response. Pulse rate, recorded at the end of the examination, was also included as a clue to autonomic response.

The measurements on all of the schizophrenic patients and on all of the non-psychotic control group were made by one person, the writer, using the following anthropometric instruments, with the technique described by Pearl (1929): anthropometer, spreading calipers, sliding calipers, steel tape line, and a manumeter.

Finally the impression of the somatic habitus of each individual was recorded. This was done by visual inspection of the nude subject at room's length, comparing mentally with an ideal average, and checking on the dual thirteen-point scale on the form sheet (the zero point of which corresponds to the extreme asthenic type and the twelve point to the extreme pyknic type) the point on each scale which seemed appropriate to her trunk and leg conformation in the examiner's judgment. This method of determining habitus type, devised by Pearl (1929), although admittedly impressionistic, is perhaps the most satisfactory method available at the present time, in that, while adhering to the terminology and general concept of habitus given by Kretschmer, it also represents habitus as a *continuous variable,* with the three *types* asthenic, intermediate, and pyknic as arbitrary triple divisions. Thus, each individual was designated one type of habitus or another by the following criteria.

Asthenic type: Trunk *and* leg scale numbers either 0, 1, 2 or 3.
Intermediate type: Trunk *and* leg scale numbers either 4, 5, 6, 7, or 8.
Pyknic type: Trunk *and* leg scale numbers either 9, 10, 11 or 12.
Dysplastic type: Trunk scale number falls into one of the above types, leg scale number into either of the other two types.

From the absolute measurements seven indices were computed for each patient and control. These were: sitting height/stature, biacromial width/stature, chest girth/stature, weight/stature, biacromial width/bicristal width, sternum length/xiphoid-symphysis length and the habitus index described by Pearl (1940), 100 (Chest girth plus abdominal girth)/stature. The use of indices is designed to avoid the subjective

factor in judgment, and to indicate to a certain extent, the somatic organization by relating two or more bodily dimensions to each other. The principal requirements for such an index of body build is discussed by Pearl (1940) whose habitus index conforms to these desiderata, is easily computed and is remarkably effective in sorting out the two extremes of body type, the asthenic and the pyknic.

ABSOLUTE ANTHROPOMETRIC MEASUREMENTS

Table 1 presents the mean, median, standard deviation, minimum observation, maximum observation, and range of variation for each of the somatic variables measured directly on each schizophrenic patient and each control individual and for the seven derivative indices. The biometric constants for right and left hand grip and for pulse rate are also shown.

The following points are to be noted:

1. The values for the total schizophrenic group are *smaller* than for the control group, and the differences are significant statistically in the following dimensions: sitting height, stature, sternum length, xiphoid-symphysis length, right and left wrist width, body weight, and right and left hand grip. The values for umbilical girth, for chest girth/stature and for the habitus index are significantly larger in the schizophrenic group than in the control group. It will be noted that the respects in which the schizophrenic patients are smaller comprise all four vertical measurements. Neither these measurements, nor those for wrist width which indicate slighter skeletal structure, are directly related to state of nutrition or age, and the differences found may be accepted as valid biological distinctions. The lighter body weight of the schizophrenic patients and the greater umbilical girth must be viewed more critically. Whereas body weight is in part related to skeletal structure, and in this respect is consistent with the findings just discussed, it is likewise markedly affected by state of nutrition and by age. Umbilical girth is likewise affected by nutrition and age, in general in a fashion paralleling weight, but in addition is also affected by the tonus of the abdominal musculature. On the basis of diet, the patients in a state hospital might be expected to fare less well than the nursing staff and the lighter average weights seem consistent with this view. On the other hand it must be recalled that the average age of the schizophrenic group is 43.5 years and of the control group 26.3 years. On the basis of age the schizophrenic group would be expected to exceed the control group in body

TABLE 1

Biometric constants of direct somatic measurements and of indices derived from direct somatic measurements of total patient group and control group

	MEAN	MEDIAN	STANDARD DEVIATION	MINIMUM OBSERVATION	MAXIMUM OBSERVATION	RANGE
Head girth						
Patients	53.7 ± .09	53.7 ± .11	1.8 ± .06	49.2	58.7	9.5
Controls	54.6 ± .39	54.6 ± .49	3.3 ± .28	52.3	57.2	4.9
Head breadth						
Patients	14.5 ±0.4	14.5 ± .50	0.9 ± .03	13.3	16.0	2.7
Controls	14.3 ± .11	14.2 ± .14	0.9 ± .08	13.7	15.0	1.3
Head length						
Patients	18.0 ± .06	18.0 ± .08	1.2 ± .04	16.2	19.6	3.4
Controls	18.4 ± .05	18.4 ± .06	0.4 ± .03	17.3	19.4	2.1
Sitting height						
Patients	81.8 ± .18	81.8 ± .22	3.7 ± .13	72.9	92.6	19.7
Controls	86.4 ± .40	86.5 ± .50	3.4 ± .29	79.2	92.4	13.2
Stature						
Patients	156.3 ± .34	156.7 ± .42	7.0 ± .24	142.9	173.4	30.5
Controls	163.1 ± .66	163.6 ± .82	5.5 ± .46	150.7	175.8	25.1
Sternum length						
Patients	15.6 ± .04	15.6 ± .05	0.9 ± .03	10.5	21.1	10.6
Controls	16.2 ± .17	16.0 ± .21	1.4 ± .12	14.0	18.6	4.6
Xiphoid-Symphysis						
Patients	32.5 ± .20	32.0 ± .25	4.1 ± .05	19.2	52.5	33.3
Controls	33.7 ± .29	33.3 ± .36	2.4 ± .20	29.4	39.7	10.3
Biacromial						
Patients	32.7 ± .11	32.6 ± .14	2.2 ± .07	24.1	39.4	15.3
Controls	33.1 ± .67	33.5 ± .86	5.6 ± .47	29.8	36.2	6.4
Bicristal						
Patients	28.3 ± .12	28.3 ± .15	2.4 ± .08	24.5	34.2	9.7
Controls	28.0 ± .15	28.2 ± .19	1.3 ± .11	24.2	30.0	5.8

TABLE 1 (*Continued*)

	MEAN	MEDIAN	STANDARD DEVIATION	MINIMUM OBSERVATION	MAXIMUM OBSERVATION	RANGE
Chest depth						
Patients	16.6 ± .06	16.3 ± .08	1.3 ± .04	12.7	22.5	9.8
Controls	16.1 ± .25	15.8 ± .31	2.1 ± .18	12.6	22.3	9.7
Wrist width—right						
Patients	4.9 ± .03	4.9 ± .04	0.6 ± .02	4.3	5.8	1.5
Controls	5.05± .04	5.0 ± .05	0.32± .27	4.6	5.5	0.9
Wrist width—left						
Patients	4.9 ± 0.1	4.9 ± .12	0.2 ± .01	4.3	5.8	1.5
Controls	5.05± .04	5.1 ± .05	0.32± .27	4.6	5.5	0.9
Chest girth						
Patients	72.8 ± .40	71.3 ± .50	8.2 ± .28	58.9	106.7	47.8
Controls	73.1 ± .63	73.4 ± .79	5.3 ± .45	61.6	90.3	28.7
Umbilical girth						
Patients	80.9 ±1.46	78.5 ± .58	9.4 ± .32	55.6	135.8	80.2
Controls	74.3 ±1.01	74.4 ±1.26	8.5 ± .72	57.7	88.4	30.7
Weight (lbs.)						
Patients	120.2 ±1.34	112.5 ±1.68	27.5 ± .94	76.0	210.0	134.0
Controls	133.0 ±2.71	135.0 ±3.39	22.0 ±1.92	93.0	186.5	93.5
Hand grip—right						
Patients	18.3 ± .34	20.0 ± .42	6.6 ± .24	5.0	40.0	35.0
Controls	32.4 ± .58	32.0 ± .72	4.9 ± .41	24.0	44.0	20.0
Hand grip—left						
Patients	16.8 ± .33	16.0 ± .41	6.4 ± .23	5.0	40.0	35.0
Controls	30.3 ± .63	30.0 ± .79	5.3 ± .45	20.0	40.0	20.0
Pulse						
Patients	92.6 ± .79	92.0 ± .99	16.2 ± .56	60.0	152.0	92.0
Controls	91.1 ±1.28	92.0 ±1.60	8.7 ± .91	76.0	108.0	32.0
Sitting height/stature						
Patients	52.3 ± .11	52.3 ± .14	2.34± .80	47.7	56.3	8.6
Controls	52.9 ± .48	52.9 ± .60	4.0 ± .34	49.5	56.4	6.9

TABLE 1 (*Continued*)

Biacromial/stature						
Patients	20.4 ± .23	20.9 ± .29	4.8 ± .16	15.9	24.9	9.0
Controls	20.3 ± .17	20.3 ± .21	1.4 ± .12	18.0	21.8	3.8
Chest girth/stature						
Patients	46.6 ± .24	45.4 ± .30	4.9 ± .17	39.1	64.0	24.9
Controls	44.8 ± .40	44.9 ± .50	3.4 ± .29	37.7	54.4	16.7
Weight/stature						
Patients	77.5 ± .82	73.0 ±1.02	16.9 ± .58	49.0	130.0	81.0
Controls	81.5 ±2.45	82.0 ±3.06	19.9 ±1.73	58.0	112.0	54.0
Biacromial/bicristal						
Patients	115.4 ± .41	116.0 ± .51	8.4 ± .29	88.0	144.0	56.0
Controls	118.0 ±1.68	119.0 ±2.11	14.1 ±1.19	105.0	133.0	28.0
Sternum length/xiphoid symphysis						
Patients	48.4 ± .43	47.0 ± .53	8.8 ± .30	29.0	90.0	61.0
Controls	48.1 ± .63	49.0 ± .79	5.3 ± .45	39.0	61.0	22.0
Habitus index						
Patients	98.3 ± .61	95.0 ± .76	12.5 ± .24	75.7	136.0	60.3
Controls	90.5 ± .83	89.3 ±1.04	7.0 ± .59	72.2	108.8	36.6

weight and umbilical girth. In the present material the lighter weight and greater umbilical girth of the schizophrenic group are probably to be accounted for by (a) a slighter skeleton and shorter height, (b) a less nutritious diet, and (c) greater atony of the abdominal and other musculature as a result of chronic illness and the inactivity of a confined existence. The greater chest girth per unit of stature and the higher value of the habitus index found in the schizophrenic group again probably reflect the operation of the age factor.

The poor showing made by the schizophrenic group in registering hand grip is in all probability attributable to a failure to arouse in the patient a sufficiently enthusiastic interest in doing her best. The results cannot be regarded as a fair test of the patient's muscular ability, but as psychomotor responses seem valuable as a quantitative indication of the degree of rapport it was possible to establish.

2. The schizophrenic group is *smaller* than the control group in the following respects, but the differences are not significant, statistically:

head girth, head length, biacromial width and chest girth, sitting height/stature, weight/stature and biacromial width/bicristal width.

3. The schizophrenic group exhibits *larger* values than the control group in the following respects, but the differences are not significant, statistically: head breadth, bicristal width, chest depth and pulse rate, biacromial width/stature, sternum length/xiphoid symphysis.

4. A comparison of the means and medians indicates that these two constants are not significantly different from each other for any of the distributions. From this it may be concluded that the distributions exhibit no asymmetry of any practical moment.

5. The range of variation between maximum and minimum values is larger in the schizophrenic group than in the control group for every dimension. The difference in range is most striking in the following respects: pulse rate, umbilical girth, weight, xiphoid-symphysis length, and chest girth. The greater range in umbilical girth, weight, and chest girth probably parallels the greater range in age (schizophrenics 54 years, controls 24 years). Xiphoid-symphysis length was found to be the most difficult to obtain accurately because of modesty reactions on the part of the patients, a fact which explains a wider range of variation in the patient than in the control group. The wider range in pulse rate in the schizophrenic group, however, is of great interest, and is probably significant of greater variability in autonomic response in the schizophrenic individual.

The smallest differences in range between the schizophrenic and control groups are found in the following measurements: wrist widths, the three head dimensions, chest depth, and bicristal width. These are all "bone" measurements which vary little with age and state of nutrition.

The differences between the schizophrenic and non-schizophrenic individuals comprising this material, in respect of their somatic dimensions, may be summarized, in broad terms, as follows: the schizophrenic individuals are significantly shorter, slighter in skeletal structure, lighter in weight (because of their smaller skeletons, less nutritious diet, and in spite of their greater mean age), larger in abdominal circumference (in spite of their lighter weight and less nutritious diet, because of their greater muscle atony from age, chronic illness and physical inactivity) and less effective in psychomotor response. The schizophrenic individuals show greater range of variation than do the controls, that in pulse rate seeming to be of particular interest as a possible indication of greater autonomic instability.

TABLE 2

Biometric constants of direct somatic measurements and of indices derived from direct somatic measurements of patients under 40 years of age and control group

	MEAN	MEDIAN	STANDARD DEVIATION	MINIMUM OBSERVATION	MAXIMUM OBSERVATION	RANGE
Head girth						
Patients	53.5 ± .23	53.4 ± .29	2.9 ± .16	49.2	57.3	8.1
Controls	54.6 ± .39	54.6 ± .49	3.3 ± .28	52.3	57.2	4.9
Head breadth						
Patients	14.3 ± .14	14.4 ± .18	1.7 ± .10	13.3	15.8	2.5
Controls	14.3 ± .11	14.2 ± .14	0.9 ± .08	13.7	15.0	1.3
Head length						
Patients	17.9 ± .14	18.0 ± .18	1.8 ± .10	16.3	19.6	3.3
Controls	18.4 ± .05	18.4 ± .06	0.4 ± .03	17.3	19.4	2.1
Sitting height						
Patients	82.2 ± .41	82.6 ± .51	5.1 ± .29	73.4	89.6	16.2
Controls	86.4 ± .40	86.5 ± .50	3.4 ± .29	79.2	92.4	13.2
Stature						
Patients	157.8 ± .43	157.7 ± .54	5.4 ± .30	145.5	173.4	27.9
Controls	163.1 ± .66	163.6 ± .85	5.5 ± .46	150.7	175.8	25.1
Sternum length						
Patients	15.5 ± .10	15.5 ± .12	1.3 ± .07	10.5	21.1	10.6
Controls	16.2 ± .17	16.0 ± .21	1.4 ± .12	14.0	18.6	4.6
Xiphoid-symphysis						
Patients	32.0 ± .02	31.6 ± .02	2.7 ± .15	25.0	40.5	15.5
Controls	33.7 ± .29	33.3 ± .36	2.4 ± .20	29.4	39.7	10.3
Biacromial						
Patients	33.0 ± .11	32.8 ± .14	1.4 ± .08	29.5	39.4	9.9
Controls	33.1 ± .67	33.5 ± .86	5.6 ± .49	29.8	36.2	6.4
Bicristal						
Patients	28.0 ± .09	27.9 ± .11	1.2 ± .07	24.7	31.3	6.6
Controls	28.0 ± .15	28.2 ± .19	1.3 ± .11	24.2	30.0	5.8

TABLE 2 (*Continued*)

	MEAN	MEDIAN	STANDARD DEVIATION	MINIMUM OBSERVATION	MAXIMUM OBSERVATION	RANGE
Chest depth						
Patients	15.8 ± .16	15.6 ± .20	1.9 ± .11	12.7	20.3	7.6
Controls	16.1 ± .25	15.8 ± .31	2.1 ± .18	12.6	22.3	9.7
Wrist width—right						
Patients	4.9 ± .06	4.9 ± .07	0.8 ± .04	4.3	5.7	1.4
Controls	5.05± .04	5.0 ± .05	0.3 ± .27	4.6	5.5	0.9
Wrist width—left						
Patients	4.9 ± .04	5.0 ± .05	0.5 ± .03	4.3	5.8	1.5
Controls	5.05± .04	5.1 ± .05	0.3 ± .27	4.6	5.5	0.9
Chest girth						
Patients	71.9 ± .51	70.6 ± .64	6.5 ± .36	61.4	91.4	30.0
Controls	73.1 ± .63	73.4 ± .79	5.3 ± .45	61.6	90.3	28.7
Umbilical girth						
Patients	76.2 ± .84	74.4 ±1.05	10.5 ± .59	55.6	110.5	54.9
Controls	74.3 ±1.01	74.4 ±1.26	8.5 ± .72	57.7	88.4	30.7
Weight (lbs.)						
Patients	116.9 ±1.68	113.5 ±2.10	20.86±1.19	87.5	169.5	82.0
Controls	133.0 ±2.71	135.0 ±3.39	22.0 ±1.92	93.0	186.5	93.5
Hand grip—right						
Patients	19.5 ± .64	20.0 ± .80	7.4 ± .45	5.0	37.0	32.0
Controls	32.4 ± .58	32.0 ± .72	4.9 ± .41	24.0	44.0	20.0
Hand grip—left						
Patients	17.5 ± .58	16.0 ± .72	6.7 ± .41	5.0	35.0	30.0
Controls	30.3 ± .63	30.0 ± .79	5.3 ± .45	20.0	40.0	20.0
Pulse						
Patients	92.4 ±1.30	92.0 ±1.62	16.3 ± .92	68.0	140.0	72.0
Controls	91.1 ±1.28	92.0 ±1.60	8.7 ± .91	76.0	108.0	32.0
Sitting height/stature						
Patients	52.1 ± .13	52.35± .16	1.68± .09	47.7	55.2	7.5
Controls	52.9 ± .48	52.9 ± .60	4.0 ± .34	49.5	56.4	6.9

TABLE 2 (*Continued*)

	MEAN	MEDIAN	STANDARD DEVIATION	MINIMUM OBSERVATION	MAXIMUM OBSERVATION	RANGE
Biacromial/stature						
Patients	20.9 ± .12	20.9 ± .15	1.53± .08	18.6	24.9	6.3
Controls	20.3 ± .17	20.3 ± .21	1.4 ± .12	18.0	21.8	3.8
Chest girth/stature						
Patients	45.6 ± .29	45.0 ± .36	3.61± .20	39.4	57.4	18.0
Controls	44.8 ± .40	44.9 ± .50	3.4 ± .29	37.7	54.4	16.7
Weight/stature						
Patients	74.0 ±1.03	72.0 ±1.35	13.4 ± .76	49.0	114.0	65.0
Controls	81.5 ±2.4	82.6 ±3.06	19.9 ±1.73	58.0	112.0	54.0
Biacromial/bicristal						
Patients	118.0 ± .69	117.0 ± .86	8.70± .49	102.0	144.0	42.0
Controls	118.0 ±1.68	119.0 ±2.11	14.1 ±1.14	105.0	133.0	28.0
Sternum length/xiphoid symphysis						
Patients	48.8 ± .63	48.0 ± .79	7.9 ±4.44	29.0	75.0	46.0
Controls	48.1 ± .63	49.0 ± .79	5.3 ± .45	39.0	61.0	22.0
Habitus index						
Patients	94.2 ± .76	91.6 ± .95	9.5 ± .53	75.7	127.7	52.0
Controls	90.5 ± .83	89.3 ±1.04	7.0 ± .59	72.2	108.8	36.6

Since the importance of age as a factor in modifying somatic habitus has been continually emphasized (Kretschmer, Henckel, Kolle, Guber-Gritz, Wertheimer and Hesketh, Perelmann and Blinkow, Burchard, Pearl and Ciocco), and since the schizophrenic group in this sample has so much greater a mean age than the control group, it seemed essential to evaluate the role which age might be playing in determining the somatic differences between the two groups. The patient group was therefore corrected for age by selecting only those individuals forty years of age or younger. A group of seventy-five schizophrenic women, with a mean age of 30.7 years was thus obtained. This group, in respect of age, corresponds reasonably to the control group with its mean age of 26.3 years.

Table 2 presents the mean, median, standard deviation, maximum observation, minimum observation and range of variation of the absolute measurements and indices for the schizophrenic group corrected for age. The biometric constants for right and left hand grip and for pulse rate are also included.

The following points are to be noted:

1. The schizophrenic patients are *smaller* than the control individuals in the following respects, and the differences are significant statistically: sitting height, stature, sternum length, xiphoid-symphysis length, right and left wrist width, weight and right and left hand grip. It will be noted immediately that these differences are identical with those found between the total schizophrenic group (uncorrected for age) and the control group. The differences in umbilical girth, in the index chest girth/stature and in the habitus index are the only ones to lose significance when the patients of younger age are used for comparison. The conclusion already drawn, namely that schizophrenic individuals *are* smaller, as indicated by height, skeletal mass and weight thus appears to be valid. The same relatively inadequate psychomotor response in the schizophrenic individuals is again noted.

2. The schizophrenic patients are *smaller* than the controls, but the differences are not significant in the following respects: head girth, head length, biacromial width, chest depth, chest girth, sitting height/stature and weight/stature. By comparison with Table 1, it will be noted that these relations are identical between the patients and controls regardless of the age factor, with the exception of chest depth which is less than the controls in the schizophrenic group corrected for age and greater in the schizophrenic group including all ages.

3. The schizophrenic patients are *larger* than the controls, but the differences are not significant, in the following respects: umbilical girth, pulse rate, biacromial width/stature, chest girth/stature, sternum length/xiphoid-symphysis, and habitus index. These relationships are also found in Table 1, but there the differences in umbilical girth and in habitus index are significant.

4. No significant differences are found between the schizophrenic and non-schizophrenic individuals in head breadth, bicristal width, and biacromial width/bicristal width, when both groups are of approximately the same average age. It will be recalled that in Table 1, head breadth and bicristal width were somewhat larger in the schizophrenic group and the index biacromial width/bicristal width somewhat smaller. A

comparison of the means and medians indicates that these two constants are not significantly different from each other for any of the distributions. From this it may be concluded that the distributions exhibit no asymmetry of any practical moment.

5. The range between maximum and minimum values is again larger for every somatic variable and every index in the schizophrenic group than in the control group, with the exception of weight, where the range for the control group is somewhat greater. The difference in range is most striking in pulse rate and umbilical girth. The remainder of the differences are relatively small, and it would thus seem that variability increases with age. The persistence of a wide range of variation in pulse rate in the younger schizophrenic patients as compared with the controls, again suggests the probable importance of this observation as a reflection of greater autonomic instability.

The differences between the schizophrenic patients corrected for age and the non-schizophrenic individuals comprising this material, in respect of their somatic dimensions, may be summarized broadly as follows: the schizophrenic patients are again found to be significantly shorter in height, slighter in skeleton, lighter in weight and less adequate in psychomotor response. The schizophrenic individuals again show, but less strikingly, greater range of variation than do the controls, that in pulse rate again standing first. It would seem that the differences between the schizophrenic patients and the controls are fundamentally valid somatic distinctions, not age-determined.

SOMATOLOGICAL COMPARISONS

Having found that differences of significance *do* exist between the schizophrenic and non-schizophrenic individuals in this material, it becomes a matter of extreme interest to see how the measurements on these two groups compare with those on other groups of individuals. In this connection Pearl and Ciocco (1934, p. 662) write: "A significant feature of constitutional somatology, as well as of physical anthropology generally, must always be intergroup comparison. In pathological matters within this field it is practically always impossible to get 'controls', in the sense in which that word is used in experimental work. Comparisons, then, of necessity, have to be made between pairs of groups differentiated from each other within broadly known limits." This question arises, in the present instance: Are these schizophrenic women of average size and the control women, perhaps, unusally large

TABLE 3

Mean stature (in centimeters) of females with various diseases

ORDER	GROUP	MEAN AGE OR AGE LIMITS *(years)*	MEAN STATURE *(cm.)*	*N*	AUTHORITY
1	Old Americans. Lab. Series. No data as to disease	Above 59	166.07	3	Hrdlička (p. 80)
2	*Non-schizophrenics*	*26.3*	*163.1 ± .66*	*32*	*This paper*
3	Old Americans. Lab. Series. No data as to disease	40-59	162.42	36	Hrdlička (p. 80)
4	Nephritis and hypertension	33.5	161.16	20	Draper (p. 334)
5	Old Americans. Lab. Series	50-59	159.18	27	Hrdlička (p. 80)
6	Tuberculosis	54.62	159.1	28	Draper (p. 334)
7	Asthma	37.72	159.04	11	Draper (p. 334)
8	Pernicious anemia	52.39	158.78	20	Draper (p. 334)
	Schizoids (Autopsy material)	59.85±1.2	157.97± .51	96	Pearl, Gooch and Freeman (p. 363)
9	*Schizophrenics*	Under 41 *(30.7)*	*157.8 ± .43*	*75*	*This paper*
	Epileptoids (Autopsy material)	56.82±3.22	157.5 ±1.68	11	Pearl, Gooch and Freeman (p. 363)
	Paranoids (Autopsy material)	70.45± .78	157.33± .51	86	Pearl, Gooch and Freeman (p. 363)
10	Non-cardiacs	49.59± .71	156.56± .83	37	Ciocco (p. 62)
11	Cardiacs	54.05± .48	156.51± .41	126	Ciocco (p. 62)
	Cycloids (Autopsy material)	65.0 ±1.97	156.53± .71	31	Pearl, Gooch and Freeman (p. 363)
12	*Schizophrenics*	*43.5*	*156.3 ± .34*	*193*	*This paper*
13	Peptic ulcers	44.1	156.21	12	Feigenbaum and Howat (p. 132)
14	Gastric and duodenal ulcers	39.17	156.0	11	Draper (p. 334)
15	Gall bladder disease	43.57	155.62	32	Draper (p. 334)
16	Diabetes	52.3	154.69	52	Feigenbaum and Howat (p. 132)
17	Schizophrenics	46.0	154.25	25	Shaw (p. 417)
18	Cholecystitis	40.8	153.92	37	Feigenbaum and Howat (p. 132)

individuals, and the differences found explained on this basis, or are the schizophrenic women *really* small as human beings go. Ciocco (1936) has assembled all of the data for the various somatic variables in the American female given in Hrdlička's *Old Americans* (weight and stature only), Draper's *Human Constitution* and in Feigenbaum and Howat's paper (1934). To these data have been added (Tables 3-11) data from Shaw's paper (1925) on somatic measurements in schizophrenic women, the only other data on the American schizophrenic female existing in the literature.

TABLE 4

Mean sitting height (in centimeters) of females with various diseases
(Age data and references same as in Table 3.)

ORDER	GROUP	MEAN SITTING HEIGHT (cm.)	N
1	*Non-schizophrenics*	*86.4 ± .40*	*32*
2	Nephritis and hypertension	84.46	20
3	Tuberculosis	84.18	28
4	Gastric and duodenal ulcers	83.83	8
5	Non-cardiacs	83.62 ± .42	37
6	Gall bladder disease	83.46	30
7	Pernicious anemia	82.78	19
8	Cardiacs	82.72 ± .23	126
9	Asthma	82.45	11
10	*Schizophrenics (40 and under)*	*82.2 ± .41*	*75*
11	*Schizophrenics*	*81.8 ± .18*	*193*

From Tables 3-11 inclusive, the following general facts appear: The schizophrenic patients are relatively short in stature (Table 3), ranking 9th, 12th and 17th in a series of eighteen compared groups, in which the control individuals of this paper rank second. The difference between the group showing the greatest mean body stature and that showing the lowest mean stature is 7.9 percent of the latter. This is a relatively small difference and indicates that stature is a relatively stable character.

The schizophrenic patients are likewise relatively short in sitting height (Table 4), ranking 10th and 11th in a series of eleven compared

TABLE 5

Mean body weight (in kg.) of females with various diseases

ORDER	GROUP	MEAN AGE OR AGE LIMITS *(years)*	MEAN BODY WEIGHT *(kg.)*	*N*	AUTHORITY
1	Gall bladder disease	43.57	72.5	26	Draper (p. 334)
2	Cardiacs	54.05±.48	68.95±.98	114	Ciocco (p. 61)
3	Old Americans. Lab. Series. No data as to disease	40-49	65.27	34	Hrdlička (p. 102)
4	Old Americans. Lab. Series. No data as to disease	Above 59	64.50	3	Hrdlička (p. 102)
5	Cholecystitis	40.8	62.87	37	Feigenbaum and Howat (p. 132)
6	Old Americans. Lab. Series. No data as to disease	50-59	62.64	26	Hrdlička (p. 102)
7	Diabetes	52.3	62.10	52	Feigenbaum and Howat (p. 132)
8	Nephritis and hypertension	33.5	60.9	20	Draper (p. 334)
9	*Non-schizophrenics.* No data as to disease	*26.3*	*60.4*	*30*	*This paper*
10	Asthma	37.72	60.4	11	Draper (p. 334)
11	Schizophrenics	46.0	57.7	25	Shaw (p. 417)
12	Non-cardiacs. No data as to disease	45.59±.71	57.06±1.3	34	Ciocco (p. 61)
13	Peptic ulcers	44.1	56.97	12	Feigenbaum and Howat (p. 132)
14	Tuberculosis	54.62	56.6	28	Draper (p. 334)
15	Gastric and duodenal ulcers	39.17	55.2	9	Draper (p. 334)
16	Pernicious anemia	52.39	54.8	20	Draper (p. 334)
17	*Schizophrenics*	*43.5*	*54.6*	*101*	*This paper*
18	*Schizophrenics* (40 and under)	*30.7*	*53.1*	*75*	*This paper*

TABLE 6

Mean chest depth (in centimeters) of females with various diseases
(Age data and references same as in Table 3.)

ORDER	GROUP	MEAN CHEST DEPTH (cm.)	N
1	Gall bladder disease	21.60	31
2	Cardiacs	21.03 ± .14	125
3	Pernicious anemia	20.91	17
4	Asthma	20.25	11
5	Nephritis and hypertension	19.98	20
6	Gastric and duodenal ulcers	19.55	11
7	Non-cardiacs	18.73 ± .28	37
8	Tuberculosis	18.55	28
9	*Schizophrenics*	*16.6 ± .06*	*192*
10	*Non-schizophrenics*	*16.1 ± .25*	*32*
11	*Schizophrenics (40 and under)*	*15.8 ± .16*	*75*

TABLE 7

Mean biacromial breadth (in centimeters) of females with various diseases
(Age data and references same as in Table 3.)

ORDER	GROUP	MEAN BIACROMIAL BREADTH (cm.)	N
1	Peptic ulcers	35.20	12
2	Nephritis and hypertension	35.12	20
3	Cardiacs	38.48 ± .12	125
4	Asthma	34.77	11
5	Gall bladder disease	34.49	30
6	Cholecystitis	33.92	37
7	Non-cardiacs	33.86 ± .22	37
8	Diabetes	33.77	52
9	Gastric and duodenal ulcers	33.75	11
10	Tuberculosis	33.61	28
11	Pernicious anemia	33.32	18
12	*Non-schizophrenics*	*33.1 ± .67*	*32*
13	*Schizophrenics (40 and under)*	*33.0 ± .11*	*75*
14	*Schizophrenics*	*32.7 ± .11*	*193*

TABLE 8

Mean biiliac breadth (in centimeters) of females with various diseases
(Age data and references same as in Table 3.)

ORDER	GROUP	MEAN BIILIAC BREADTH *(cm.)*	*N*
1	Cardiacs	29.44 ± .14	123
2	Pernicious anemia	29.35	18
3	Gall bladder disease	29.33	30
4	Gastric and duodenal ulcers	28.65	11
5	Tuberculosis	28.52	27
6	Nephritis and hypertension	28.46	20
7	*Schizophrenics*	*28.3 ± .12*	*192*
8	Asthma	28.20	11
9	Non-cardiacs	28.03 ± .25	37
10	*Non-schizophrenics*	*28.0 ± .15*	*32*
11	*Schizophrenics (40 and under)*	*28.0 ± .09*	*75*

TABLE 9

Mean head circumference (in centimeters) of females with various diseases
(Age data and references same as in Table 3.)

ORDER	GROUP	MEAN HEAD CIRCUMFERENCE *(cm.)*	*N*
1	Gastric and duodenal ulcers	55.06	7
2	Asthma	54.87	3
3	Cardiacs	54.84 ± .09	123
4	Nephritis and hypertension	54.70	6
5	*Non-schizophrenics*	*54.6 ± .39*	*32*
6	Non-cardiacs	54.58 ± .19	37
7	Gall bladder disease	54.12	29
8	Tuberculosis	54.04	26
9	*Schizophrenics*	*53.7 ± .09*	*193*
10	*Schizophrenics (40 and under)*	*53.5 ±*	*75*

TABLE 10

Mean head length (in centimeters) of females with various diseases
(Age data and references same as in Table 9.)

ORDER	GROUP	MEAN HEAD LENGTH *(cm.)*	*N*
1	Nephritis and hypertension.....	18.50	20
2	Asthma	18.48	11
3	*Non-schizophrenics*	*18.4 ± .05*	*32*
4	Cardiacs	18.36 ± .04	125
5	Gall bladder disease.....	18.23	32
6	Tuberculosis	18.22	28
7	Gastric and duodenal ulcers.....	18.21	32
8	Pernicious anemia	18.11	21
9	*Schizophrenics*	*18.0 ± .06*	*193*
10	Non-cardiacs	17.99 ± .09	37
11	*Schizophrenics (40 and under)*.....	*17.9 ± .14*	*75*
12	Peptic ulcers	17.40	12
13	Diabetes	17.31	52
14	Cholecystitis	17.25	37

groups in which the control group of this paper ranks first. The difference between the group showing the greatest mean sitting height and that showing the lowest is 5.6 percent of the latter, again a relatively small difference.

In the case of body weight (Table 5), the schizophrenic patients are again at the bottom of the list, ranking 11th, 17th, and 18th in a series of eighteen compared groups in which the control group of this paper ranks 9th. The difference between the group showing the greatest mean weight and that showing the lowest is 36.5 percent of the latter, a large difference which reflects the relatively greater variability of weight as a constitutional character.

Table 6, showing chest depth, reveals the schizophrenic patients 9th and 11th in a series of eleven compared groups. The non-schizophrenic group of this paper ranks 10th. This extremely low position can probably be accounted for, in part, by the much younger mean age of this control group (26.3 years) as compared with the mean ages for the groups ranking first through eighth of this table. The difference between

TABLE 11

Mean head breadth (in centimeters) of females with various diseases
(Age data and references same as in Table 9.)

ORDER	GROUP	MEAN HEAD BREADTH *(cm.)*	*N*
1	Pernicious anemia	14.91	21
2	Gall bladder disease	14.88	32
3	Cardiacs	14.88 ± .04	125
4	Asthma	14.78	11
5	Nephritis and hypertension	14.65	19
6	Gastric and duodenal ulcers	14.64	12
7	Tuberculosis	14.50	28
8	*Schizophrenics*	*14.5 ± .40*	*193*
9	Diabetes	14.34	52
10	*Non-schizophrenics*	*14.3 ± .11*	*32*
11	*Schizophrenics (40 and under)*	*14.3 ± .14*	*75*
12	Non-cardiacs	14.28 ± .08	37
13	Cholecystitis	14.23	37
14	Peptic ulcers	13.95	12

the groups with largest and lowest mean values for chest depth is 36.7 percent of the latter, a large difference indicating the variability of this character.

In Table 7, showing biacromial width, the schizophrenic patients again appear at the end of the list, ranking 13th and 14th in a series of fourteen compared groups. The control group of this paper ranks 12th, but the differences between the control mean and that of the preceding six groups is slight. This is borne out by the fact that the difference between the groups with largest and lowest mean values for biacromial width is only 7.6 percent of the latter.

Table 8, showing bicristal width, again reveals the schizophrenic patients relatively low on the list, ranking 7th and 11th in a series of eleven compared groups. However, the difference between the largest and lowest means is only 1.4 cms., or 5.0 percent of the value of the lowest mean.

In Tables 9, 10, and 11, showing head measurements, the schizophrenic patients continue to appear in the lower range of the mean values.

They rank 9th and 10th in head circumference in a series of ten compared measurements in which the controls rank 5th. In head length, the schizophrenic group ranks 9th and 11th in a series of fourteen compared measurements in which the controls rank third. In head breadth, the schizophrenics rank 8th and 11th in a series of fourteen compared measurements, the controls standing 10th. The differences between the greatest and least mean values for these head measurements is slight, but the persistent finding of relatively small values for the schizophrenic patients is interesting.

Reviewing these tables (3-11) as a whole, it may be noted that in no table is the schizophrenic group found in the upper third of the series, and in only one table (Table 3) is the schizophrenic group found higher than the upper half of the series (here the schizophrenic group forty years of age and under of this paper ranks 9th in a series of eighteen). A schizophrenic group is found in the last third of every table. The control group, on the other hand, does not fall consistently in the same relative ranking from table to table, but is found represented evenly in upper, middle, and lower thirds of the series. Differences between the schizophrenic and control groups in this paper are not, therefore, due to any unique and atypical selection of somatic type in the control individuals, but may be interpreted as true deviations from a fairly adequate control norm.

One other pertinent set of measurements may be mentioned, that of Pearl, Gooch, and Freeman (1935), not strictly comparable because obtained on autopsy material. They found the mean stature of 96 female schizoids whose mean age at death was 59.85±1.1 years to be 157.97±.51 cms.; of 86 paranoids with a mean age at death of 70.45±.78 years to be 157.33±.51 cms.; of 11 epileptoids whose mean age at death was 56.82±3.2 years to be 157.5±1.68 cms.; and of 31 cycloids whose mean age at death was 65.0±1.97 years to be 156.3±.34 cms. These results correspond very closely to those found in the present study in the patient group and are interesting in relation to the series shown in Table 3.

While the number of actual measurements on schizophrenic females recorded in the literature is scarce, more data on male schizophrenics can be found. These data on weight and height for seven groups of schizophrenic males are presented in Tables 12 and 13, and the position of the mean value for each group in its relation to the mean values for sixty-six groups, other than schizophrenic, compiled by Pearl and Ciocco in

1934. The groups with the highest and lowest mean values for each somatic variable in Pearl and Ciocco's tables are included. In Table 12 it will be noted that, in respect of weight, schizophrenic males rank 38th, 51st, 60th, 65th, 69th, 70th, and 72nd in a series of twenty-three compared groups. That is, all of the schizophrenic groups fall in the lower half of the series and all but one (Wells) fall in the lowest third of the series. Similarly, in Table 13, the schizophrenic groups rank 62nd, 65th, 67th, 68th, 70th, 72nd and 73rd in a series of seventy-three compared measurements. That is, all of the schizophrenic groups fall in the lowest one-fifth of the series. These results for schizophrenic males are in complete accord with the findings for schizophrenic females shown in Tables 3-11.

From these tables showing the relative position of male and female schizophrenic patients as compared with non-schizophrenic individuals in respect of various somatic variables, the impression that the schizophrenic individuals are, in fact, small people seems corroborated. A

TABLE 12

Mean weight (in kgs.) of schizophrenic males, in relation to males with various diseases as compiled from the literature by Pearl and Ciocco (1934, pp. 663-669)

ORDER	GROUP	MEAN WEIGHT (kg.)	*N*	AUTHORITY
1	U.S. Army officers with degenerative cardio-vascular-renal disease 10 years after measurement	79.11 ± .78	108	Reed and Love
38	Schizophrenia	65.8	50	Wells
51	Schizophrenia	63.2	100	Kolle (Jena)
60	Schizophrenia	62.5	100	Kolle (Schwein)
65	Schizophrenia	62.01	125	Burchard
69	Schizophrenia	58.3	100	Henckel
70	Schizophrenia	57.7	25	Shaw
72	Schizophrenia	54.1	100	Matecki and Szpidbaum
73	Draft recruits, underweight	50.32	12,129	Davenport and Love

TABLE 13

Mean height (in cms.) of schizophrenic males, in relation to males with various diseases as compiled from the literature by Pearl and Ciocco (1934, pp. 670-676)

ORDER	GROUP	MEAN HEIGHT (cm.)	*N*	AUTHORITY
1	U.S. Army officers with valvular heart disease (other than aortic regurgitation) at age measured	177.52 ± .84	19	Reed and Love
62	Schizophrenia	169.45	125	Burchard
65	Schizophrenia	168.7	100	Kolle (Schwein)
67	Schizophrenia	167.6	150	Wells
68	Schizophrenia	167.0	100	Henckel
70	Schizophrenia	165.3	100	Kolle (Jena)
71	Gastric and duodenal ulcer	164.28	30	Draper
72	Schizophrenia	160.5	100	Matecki and Szpidbaum
73	Schizophrenia	160.2	25	Shaw

quantitatively limited endowment of soma would seem to be a handicap of significance, so far as the capacity of the personality for resisting a schizophrenic reaction is concerned. The next point in question is how this somatic endowment, in the schizophrenic as compared with the non-schizophrenic individuals, is *organized* into the somatic unit which represents the *person*.

(*To be concluded*)

INTRACRANIAL VARIATION IN THE WEIGHT OF THE HUMAN BRAIN*

BY F. W. APPEL AND E. M. APPEL

INTRODUCTION

SINCE 1905 it has been evident that the weight of the human brain varies significantly with race, sex, age, some dimensions of the head and of the body, and body weight. Pearl's analysis in that year of the fairly large quantities of reliable data collected by Bischoff (1880), Retzius (1900), Marchand (1902), and Matiegka (1902) showed that this variation can be determined and expressed exactly. But it showed, too, that much more comprehensive data are needed if the full significance of the variation is to be realized.

A new aspect of the variation is emphasized in the present work, namely, the variation which occurs within the skull, presumably in heads of all morphological types. To make this aspect clear the material on which we worked must be described, for certain characteristics of the material itself are responsible for the direction which the study took.

The material came from the St. Elizabeth's Hospital of Washington, D. C. Dr. Walter Freeman, who was formerly pathologist at this hospital, brought certain biometric data from the autopsy records of the Hospital to the Department of Biology of the Johns Hopkins School of Hygiene and Public Health for statistical analysis. Beginning with the first autopsy after the founding of the Hospital, the records date from 1884. The records available for the present work run consecutively through 1924, and then skip to a few for 1933. The cases for the interval between 1924 and 1933 were used by Freeman in his studies on the endocrine organs (Freeman, 1931, 1934; Pearl, Gooch, and Freeman,

* The material for this work was placed at the disposal of the authors by Dr. Raymond Pearl, who offered not only the facilities of his statistical laboratory but also his personal help in devising fruitful ways of getting at whatever might lie hidden in the data. For his kindness, his invaluable guidance, and his collaboration, the authors are sincerely grateful. Dr. Pearl did not live to see the conclusion of the work, the responsibility for which is therefore solely our own.

1935). For the remaining body of material, totaling nearly 4500 data sheets, 2752 records have been segregated. These include all of the *male, white* cases for which the weight of the brain is given. This array of cases is much more extensive than any series for one sex and one race which has been available heretofore from a single source.

No racial subdivision of this group of cases is possible. Diverse European stocks are undoubtedly represented and possibly there is some admixture of non-European stock. The patients came to St. Elizabeth's Hospital from the civil and military services of the United States government. Presumably they came from all sections of America.

Weights are available for several organs besides the brain, particularly the heart, the spleen, the kidneys, and the liver. Unfortunately neither the weight of the body nor any of its dimensions are given. Correlative information is available, however, on the length and breadth of the skull, the causes of death, the age at death, the year of death, and the lapse of time between death and autopsy.

The information on this last-named point is especially noteworthy. Since all that is known about the weight of the brain comes from post-mortem measurement, the possibility of post-mortem change in weight needs to be thoroughly explored; and this has never been done.

Nothing is known about the conditions under which the organs were measured, except what Freeman has to say about his own procedures (1931). Only a fraction of the autopsies were done under Freeman's term of office. The post-mortem examinations were done by many different people, working at different periods in the history of the Hospital. Nearly 50 years are covered by the records. Brandes (1927) states that the dura mater weighs 50-60 grams, and that the complete drainage of the cerebrospinal fluid from the fresh brain may change its weight by as much as 50 grams. We presume that in all autopsies at St. Elizabeth's Hospital the outer meninx was removed from the brain insofar as it could be, but that the inner meninges, the arachnoid and pia layers, were not. We presume, too, that the brains were weighed immediately after removal, and just as they came from the skulls (that is, without being either rinsed or drained), but we do not know this. Certainly considerable differences in the weights of brains might result from different modes of handling.

Most studies on the weight of the brain have been based on hospitalized populations — populations composed mainly of persons who were ill with acute or chronic diseases, or who required surgical

treatment, and who died while being treated for these conditions. The patients who came to autopsy at St. Elizabeth's Hospital had a wide assortment of bodily ills. But they were an especially selected group in that they were either mentally ill or suspected of being so. It would be a mistake to place too much emphasis on this point. Most of the information available about the weight of the human brain has come from mental hospitals and pathological institutions. Nevertheless the special selection of the present material should be kept in mind, and later it will be considered again.

Most of the men whose deaths furnished the basis for this study died at advanced ages. Many of them lived for years in the Hospital before they died, and many of them died of diseases which are more commonly encountered in older men than in the young. No doubt the age distribution of the cases is one which might be expected in the autopsy population of a mental hospital. In respect to it, the selected character of the material has proved to be peculiarly favorable to our study.

This paper is an initial survey. It deal especially with the variation of the brain weight with skull dimensions, age at death, and time post-mortem. Some consideration has been given to the conditions of diseases as represented by the causes of death.

For reasons which will be apparent, it seemed desirable to distinguish between organs to which any suspicion of abnormality could be attached because of lesion or malformation or disease, and organs which were entirely free from any recognized disease or abnormality of form. Weight *per se* could not be taken as a criterion, of course, but the records provide many indications as to the normality of the organs, some by explicit statements in the pathological reports, some by implications in the statements as to the causes of death. An attempt was made to classify each organ as either (1) non-lesioned, or (2) lesioned. All of the brains could be classified definitely one way or the other. In a very few instances where no weight was given for the heart, liver, spleen, or kidneys, judgment was suspended as to the normality of these organs. The present paper has to do only with the brain weights.

Because of the number of cases and the number of items of information about them, the data for this initial survey were assembled and assorted mechanically. A numerical code was drawn up for the non-numerical items, so that the available information could be transferred in numerical form to punchcards. By means of a machine these

cards were then sorted into classes and subclasses according to each item of information that was wanted from them. After each sorting the number of cards in each class or subclass, together with certain maximal and minimal values for the class, were recorded on tabular forms for subsequent analysis. Only the information derived from the basic tables, and only a part of that, can be presented here.

In all of the work which follows the brain weights were grouped in 100-gram classes. Smaller groupings would not materially change our findings, but they would be advisable in subsequent work. The probable error of each weight is given with it.

THE FUNDAMENTAL CONSTANTS

Of the 2752 brains only 672 appear to have any suspicion of abnormality attached to them because of lesions, malformations, or disease. No physical abnormalities were recognized at autopsy in the brains of the remaining 2080 cases. Since the cases came from a mental hospital, such a high proportion of non-lesioned and *apparently* normal brains was not anticipated. Interest centers in this non-lesioned group.

The constants for weight for both groups and for the combined array are:

	Non-lesioned	*Lesioned*	*Total*
Number of cases.....	2080 (= 75.6 percent)	672 (= 24.4 percent)	2752 (= 100 percent)
Range (grams).....	870–1750	724–1827	724–1827
Mean (grams).....	1305.29 ± 1.92	1287.65 ± 3.97	1300.98 ± 1.75
Median (grams).....	1301.67 ± 2.41	1281.42 ± 4.97	1296.88 ± 2.19
Standard Deviation (grams)	129.96 ± 1.36	152.45 ± 2.81	136.00 ± 1.24
Coefficient of Variation (percent)	9.96 ± 0.11	11.84 ± 0.22	10.46 ± 0.10

The constants are substantially the same for the non-lesioned cases and for the total array. The means differ by only 4.3 ± 2.6 grams. The mean for the lesioned group differs by 17.6±4.4 grams (1.35 percent) from the mean for the non-lesioned group; the median differs by 20.3±5.5 grams (1.56 percent). The weights are widely scattered — in each group the highest weights are more than double the lowest. As might be expected, the weights are more variable in the lesioned group than the non-lesioned.

For the non-lesioned group the standard deviation is nearly 130 grams, or 10 percent of the mean. This is noteworthy, because it

places a definite limitation upon the certainty of the significance of the small deviations from this mean which will be set forth in the work which follows. It is well to remember, however, that deviations of less than 50 grams may be biologically significant, or at least suggestive, in spite of being statistically inconclusive.

In all but one of the 2752 cases the age at death is given. The basic constants for age are as follows:

	Non-lesioned	*Lesioned*
Number of cases	2079	672
Range (years)	12–96	19–95
Mean (years)	59.93 ± 0.24	67.11 ± 0.36
Median (years)	62.70 ± 0.30	67.36 ± 0.45
Standard Deviation (years)	15.95 ± 0.17	13.32 ± 0.25
Coefficient of Variation (percent)	26.61 ± 0.29	19.85 ± 0.38

Between the non-lesioned and the lesioned groups there is a mean age difference of 7.18±0.43 years. This difference is more than 17 times its probable error. In both groups the ages are extremely variable; indeed, they are much more so than the weights of the brains.

In the work which follows the non-lesioned-brain cases alone will be used.

VARIATION OF BRAIN WEIGHT WITH SKULL CHARACTERISTICS

Only about a third of the data sheets for the non-lesioned-brain cases provide measurements of the length and breadth of the skull. Table I shows how the weight of the brain varies with these skull dimensions and with the cephalic index.

The dimensions of the skull are correlated positively with the weight of the brain. The coefficient of correlation is +.355±.021 for skull length, and +.305±.022 for skull breadth. The correlation ratios are .350 and .287, respectively. These ratios do not differ significantly from the correlation coefficients, hence the regressions of brain weight on skull length and skull breadth do not deviate significantly from being linear. The regression equations are:

$$\text{Brain weight (in grams)} = 186.40 + 5.945\ L$$
$$\text{Brain weight (in grams)} = 354.39 + 6.406\ B$$

Here L is the skull length, B the skull breadth, in millimeters. The regression lines calculated means of these equations are shown with the observed mean weights in Figs. 1 and 2.

TABLE 1

Variation in brain weight with skull characteristics
(Non-lesioned-brain cases)

	NO. CASES	RANGE *(grams)*	MEAN *(grams)*	MEDIAN *(grams)*	STANDARD DEVIATION *(grams)*	COEFFICIENT OF VARIATION *(percent)*
Skull length *(mm.)*						
146–169	10	1060–1318	1220.00±20.53	1240.00±25.73	96.26±14.52	7.89±1.20
170–179	155	936–1616	1236.45± 5.86	1227.00± 7.34	108.13± 4.14	8.75± .34
180–189	360	950–1720	1284.17± 4.46	1282.41± 5.59	125.50± 3.15	9.78± .25
190–199	207	1020–1700	1334.54± 5.66	1331.62± 7.09	120.75± 4.00	9.05± .30
200–216	26	1148–1729	1438.46±21.47	1442.86±26.91	162.30±15.18	11.29±1.07
Total	758		1292.61± 3.18			
Skull breadth *(mm.)*						
127–129	7	936–1375	1192.86±34.92	1225.00±43.76	136.96±24.69	11.49±2.10
130–139	111	1000–1478	1235.86± 6.33	1234.62± 7.93	98.84± 4.47	8.00± .36
140–149	422	950–1616	1284.83± 4.04	1285.00± 5.06	123.02± 2.86	9.58± .22
150–159	199	971–1729	1334.92± 6.47	1322.73± 8.11	135.41± 4.58	10.15± .35
160–171	18	1091–1720	1400.00±26.23	1416.67±32.88	164.99±18.55	11.79±1.34
Total	757		1292.67± 3.18			
Cephalic indices						
64.2–69.9	11	1117–1424	1295.45±17.14	1310.00±21.48	84.26±12.12	6.51±0.94
70.0–74.9	162	936–1634	1278.40± 6.93	1269.81± 8.69	130.87± 4.90	10.24± .39
75.0–79.9	317	972–1729	1305.84± 4.97	1307.81± 6.23	131.21± 3.52	10.05± .27
80.0–84.9	183	950–1680	1287.70± 6.30	1285.00± 7.89	126.26± 4.45	9.81± .35
85.0–89.9	75	1050–1616	1272.67± 9.38	1256.45±11.76	120.50± 6.64	9.47± .53
90.0–95.9	9	1060–1720	1350.00±40.53	1315.00±50.80	180.28±28.66	13.36±2.16
Total	757		1292.67± 3.18			

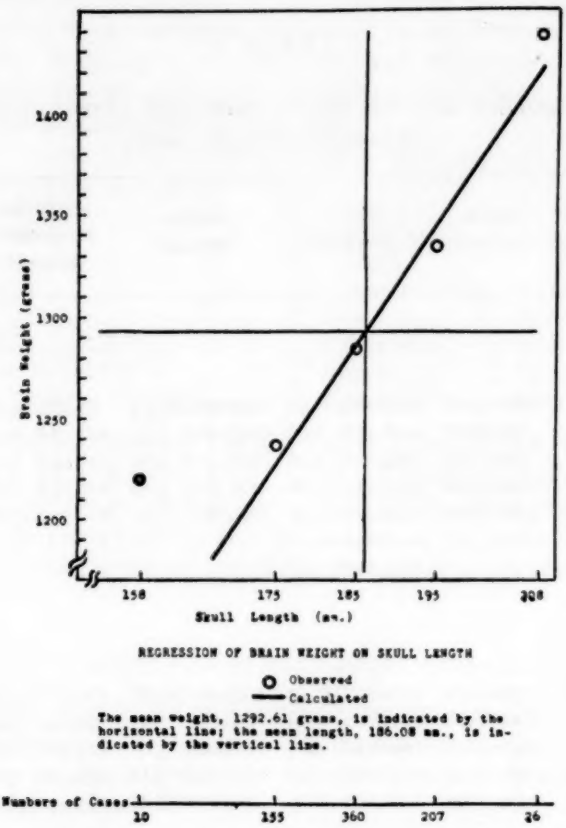


FIG. 1

Our findings differ markedly from those of Pearl (1905) in the absolute values of the observed mean weights for each class of skull lengths and skull breadths, but they do not differ in demonstrating that the weight increases with length or breadth. Pearl found somewhat higher coefficients of correlation for one of his arrays of cases (for Bohemian males the coefficient is $+.548\pm.027$ for skull length, and $+.466\pm.031$ for skull breadth). Insofar as the brain weight is determined by the size of the skull, this comparison would seem to show that Pearl's material was more homogeneous in head size than ours. But we have only two skull dimensions to serve as indices of head

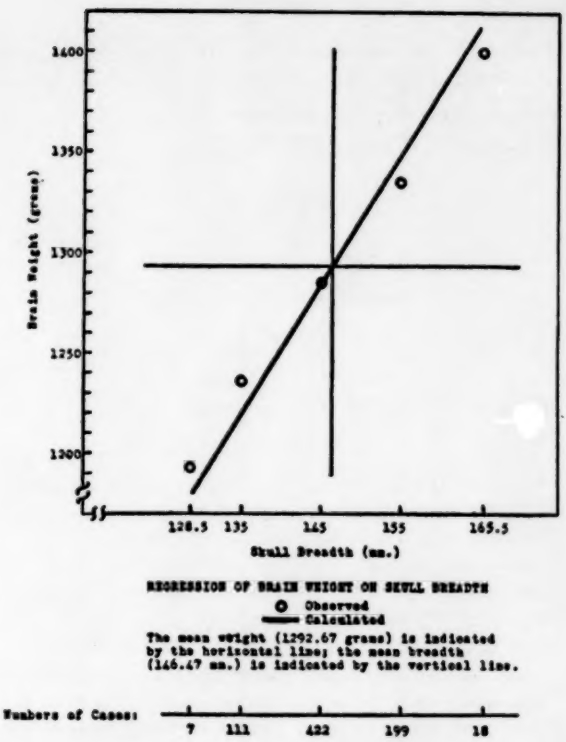


FIG. 2

size; and furthermore, as we shall show, the brain does not vary in weight solely with the size of the skull.

The cephalic index, the ratio of the skull breadth to the skull length, in percent, is a measure of the shape of the skull. For our material the cephalic indices are highly variable, indicating that heterogeneous types of head shapes are represented. The brain weight varies quite irregularly with the cephalic indices. Unfortunately these indices cannot be used as criteria of head size, for heads that are very different in size may have the same cephalic index. For instance, a long broad skull has the same cephalic index as a short narrow one, if the ratio of breadth to length is the same. The skull dimensions provide our only means of estimating the relation of the brain weight to the size of the cranium.

VARIATION IN BRAIN WEIGHT WITH AGE AT DEATH

The array of cases is remarkable for the numbers of individuals in the older ages. For the ages beyond 25 years there are enough cases so that the variation of the brain weight can be studied by 5-year age-groups. The variation constants are given in Table 2.

TABLE 2

Variation in brain weight with age
(Non-lesioned-brain cases)

AGE CLASSES *(years)*	NO. CASES	RANGE *(grams)*	MEAN *(grams)*	MEDIAN *(grams)*	STANDARD DEVIATION *(grams)*	COEFFICIENT OF VARIATION *(percent)*
12–24	19	870–1700	1286.84±27.23	1307.14±34.13	175.98±19.26	13.68±1.52
25–29	61	1050–1600	1361.48±12.37	1380.77±15.50	143.21± 8.75	10.52±0.65
30–34	105	1060–1660	1375.71± 8.41	1371.62±10.54	127.73± 5.94	9.29±0.44
35–39	109	1050–1634	1333.49± 8.57	1325.00±10.74	132.70± 6.06	9.96±0.46
40–44	141	936–1750	1338.65± 7.66	1335.00± 9.60	134.86± 5.42	10.08±0.41
45–49	151	1020–1729	1327.48± 7.52	1314.10± 9.43	137.08± 5.32	10.33±0.41
50–54	182	1046–1720	1326.92± 6.43	1327.12± 8.06	128.63± 4.55	9.70±0.35
55–59	164	990–1720	1301.83± 6.77	1293.75± 8.49	128.61± 4.79	9.88±0.37
60–64	199	950–1690	1306.78± 5.97	1312.88± 7.48	124.86± 4.22	9.56±0.33
65–69	257	1000–1700	1309.53± 5.44	1306.08± 6.82	129.43± 3.85	9.89±0.30
70–74	303	972–1620	1283.00± 4.64	1280.73± 5.81	119.65± 3.28	9.33±0.26
75–79	223	934–1550	1258.97± 4.89	1256.55± 6.13	108.31± 3.46	8.61±0.28
80–84	126	971–1560	1253.97± 6.48	1251.11± 8.12	107.76± 4.58	8.60±0.37
85–96	39	1050–1400	1232.05±11.00	1229.17±13.79	101.90± 7.78	8.27±0.64
Not given	1					
25–96	2060*		1305.44± 1.92		129.47± 1.36	
Total	2080		1305.29± 1.92		129.96± 1.36	

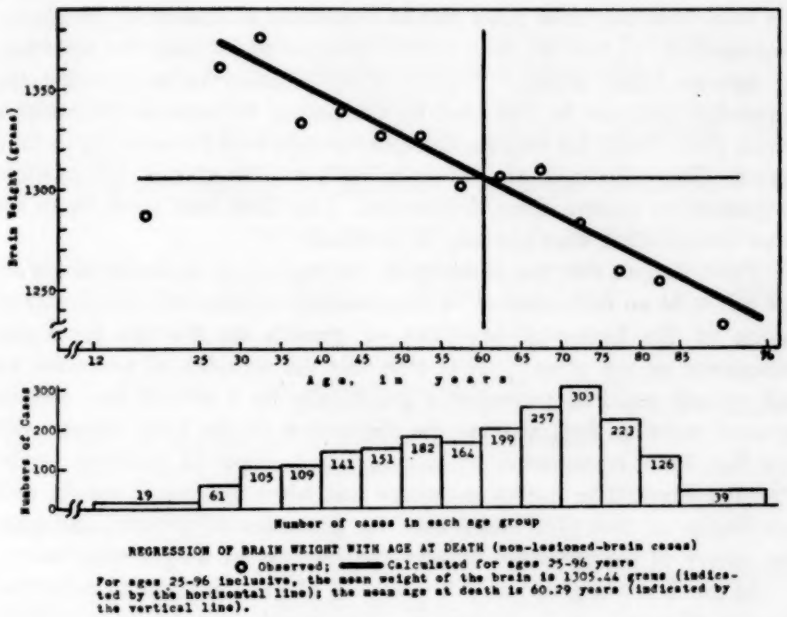
* These are the cases used in calculating the regression of brain weight with age. See Fig. 3.

Within each age group the weights vary over a wide range; the standard deviations are relatively high. In spite of the variability a definite trend can be seen. The mean weight is greatest at some age in early manhood; it decreases thereafter with advancing age. For ages 25 through 96 the coefficient of correlation between weight and age is

—.245±.014, and the correlation ratio is .258. The difference between the squares of these indices is insignificant (it is only .0062±.0023), hence the regression of weight on age is linear, between these ages at least. The regression equation is:

$$\text{Brain weight (in grams)} = 1428.48 - 2.041\,A$$

where A is the age in years. The slope of the regression line is shown in Fig. 3, which shows also the observed mean weights for the successive age groups.



REGRESSION OF BRAIN WEIGHT WITH AGE AT DEATH (non-lesioned-brain cases)

○ Observed; ——— Calculated for ages 25-96 years

For ages 25-96 inclusive, the mean weight of the brain is 1305.44 grams (indicated by the horizontal line); the mean age at death is 60.29 years (indicated by the vertical line).

FIG. 3

The trend of the mean weight with advancing age has been known for many years (see Pearl, 1905). Pearl's regressions are of practically the same slope as ours. Pearl found a coefficient of correlation of —.249±.031 for his best array of male data, and a correlation ratio of .288. Pearl judged that the decline in weight must be continuous and at a uniform rate throughout adult life, but he maintained some reserve in drawing this inference, especially for the older ages, because the available evidence was statistically inconclusive. On this point it seems

likely that the present array of cases is extensive enough to be conclusive. The loss in weight seems to take place at a rate which is remarkably uniform, considering the variability of the weights in other respects, and there is no reason to suppose that the rate of decrease changes at any age, however advanced. It must be pointed out, however, that we do not yet know the extent to which our estimate of this weight regression must be modified to allow for the effects of other factors of variability. Adjustments for skull size are not likely to change the relation between age and brain weight appreciably, though they could do so, of course: but we have made no allowance as yet for any swelling or shrinking of the brain that may take place due to conditions of disease or conditions post-mortem. Later we may be in a position to estimate the net effect of age on brain weight. The need for caution in interpreting the foregoing data can be indicated by comparing the age-specific weights which Pearl found for various European groups with those which we have found. The differences are shown in Table 3. No attempt will be made at present to resolve these differences. Our first task must be to see what factors other than age may be involved.

Pearl judged that the linearity of the regression of brain weight on age might be an indication of "a fundamental difference in the modes of action of the biological processes of growth on the one hand and senescence on the other." It is true that the complex of processes we call growth must be represented graphically by a curved line, and an upward curve at that, whereas the diminution of the brain weight with age has been represented by a downward slope of uniform grade. Perhaps it would be useful to inquire just when the loss in weight with age begins to take precedence over the processes of growth, and what the nature of the processes involved in the loss of weight may be.

In the first place, certainly it would be of great interest to determine the age of maximum brain weight. Presumably this age is as variable as the age of the attainment of physical maturity in any other bodily feature, for instance stature, or head size. In our material the decrease in brain weight seems to begin between age 20 and age 30. Pearl was inclined to believe that the decrease might begin between the 15th and 20th years of life, because the regression of weight on age seemed to be linear as early as age 15 (Hessian data) or age 20 (Swedish data), so far as could be judged. Nor does the regression deviate much from being linear in our data when ages under 25 are included in the calculation. We have very few cases for the earlier ages, however:

TABLE 3

Comparison of constants

	AMERICANS [1]	BAVARIANS [2]	BOHEMIANS [3]	HESSIANS [4]	SWEDES [5]
	I. *Constants for brain weight, white males*				
Number of cases	2080	529	372	475	416
Mean (grams)	1305.29±1.92	1363.19±3.25	1454.84±3.97	1391.74±3.49	1400.48±3.52
Standard Deviation (grams)	129.96±1.36	110.64±2.30	113.61±2.81	112.68±2.47	106.33±2.49
Coefficient of Variation (percent)	9.96±0.11	8.12±0.12	7.81±0.20	8.10±0.18	7.59±0.18
	II. *Constants for age at death, white males*				
Number of cases	2079	529	372	475	416
Mean (years)..	59.93±0.24	43.17±0.40	45.70±0.52	42.89±0.52	45.02±0.45
Standard Deviation (years)	15.95±0.17	13.50±0.28	14.96±0.37	16.84±0.37	13.67±0.32

III. *Mean brain weight (in grams) by age at death*

AGE	AMERICANS		BAVARIANS		HESSIANS		SWEDES		BOHEMIANS		AGE
(years)	*No.*	*Weight*	*No.*	*Weight*	*No.*	*Weight*	*No.*	*Weight*	*No.*	*Weight*	*(years)*
15–19	4	1350±22	..	.....	36	1407±10	..	.....	..	.....	.....
20–24	14	1300±29	32	1361±10	58	1431±10	27	1432±12	77	1388±4	20–29
25–29	61	1361±12	54	1375± 9	52	1398±10	37	1453±12			
30–34	105	1376± 8	78	1378± 9	41	1408±12	48	1435±11	58	1379±6	30–39
35–39	109	1333± 9	104	1361± 8	24	1371±14	49	1404±11			
40–44	141	1339± 8	52	1374± 9	42	1399±11	61	1382±10	91	1337±4	40–49
45–49	151	1327± 8	45	1366±10	38	1404±14	40	1410± 9			
50–54	182	1327± 6	58	1378± 9	55	1370±11	50	1404± 8	77	1356±4	50–59
55–59	164	1302± 7	37	1368±13	41	1380±10	39	1375±10			
60–64	199	1307± 6	27	1349±15	33	1373±17	32	1359±13	46	1339±6	60–69
65–69	257	1310± 5	19	1304±15	30	1370±13	18	1344±15			
70–74	303	1283± 5	15	1305±17	17	1351±20	6	1342±12	23	1280±3	70–79
75–79	223	1259± 5	8	1263±29	8	1331±12	9	1358±16			

[1] St. Elizabeth's Hospital cases.
[2] Pearl's material; original data from Bischoff, 1880.
[3] Pearl's material; original data from Matiegka, 1902.
[4] Pearl's material; original data from Marchand, 1902.
[5] Pearl's material; original data from Retzius, 1900.

only 14 for the 20—24-year class, only 4 for the 15—19-year class, and only 1 case for an earlier age. To us it seems not incredible that the reduction in the size of the brain should begin as early as age 15, but the age of maximum brain weight could only be determined if we had at hand a considerable body of data for the early years of life as well as the later ones.

In 1910 and in 1916 Donaldson presented evidence that the percentage of water in the brain of the white rat decreases with the age of the animal. This is not true of the weight of the brain, however; the weight increases with the age of the rat. Donaldson found that the heavier brains contained not only less water but more myelin, so he suggested that the loss in weight due to loss of water may be more than offset by the gain in weight due to myelination. Presumably in man as well as in the rat a loss of water occurs with age; but quantitative evidence on this point would be very useful in determining how much of the loss in weight should be attributed to water loss, if any.

Andrew reported in 1939 that a process of "neuronophagia" takes place in the cerebral cortex in man as a result of the normal aging process. The large pyramidal cells of the cortex are ingested by their satellite glia cells. With advancing age an increase takes place in the number of satellite cells. How this cellular increase may be related to changes in the fluid environment of the brain we do not know, nor is it clear how it can be reconciled with the decrease in weight which Pearl found, and which we also found.

Probably it is safe to assume that the change in brain weight with age is not due simply to dehydration, though dehydration may have something to do with it.

Let us assume for the moment that the regression we have found for weight on age is the net or true regression for our array of cases. In order to estimate the magnitude of the over-all loss in weight with age, let us suppose further that the age of maximum weight is 25, and that life continues to age 96. The over-all loss in mean weight would then be $1377 - 1233 = 144$ grams. This is about 11 percent of the mean weight for the total array of non-lesioned-brain cases. The maximum loss as thus estimated may be far from the true loss even for this series of cases. Still, the estimate gives us a figure which may be compared with the maximum gain that occurs after death.

VARIATION IN BRAIN WEIGHT WITH HOURS POST MORTEM

For more than 74 percent of the non-lesioned-brain cases, namely for 1544 out of 2080, the numbers of hours elapsing between death and autopsy have been recorded. The interval varies from 1 hour to 146. Only 3 cases were examined within 1 hour of death, 20 within the second

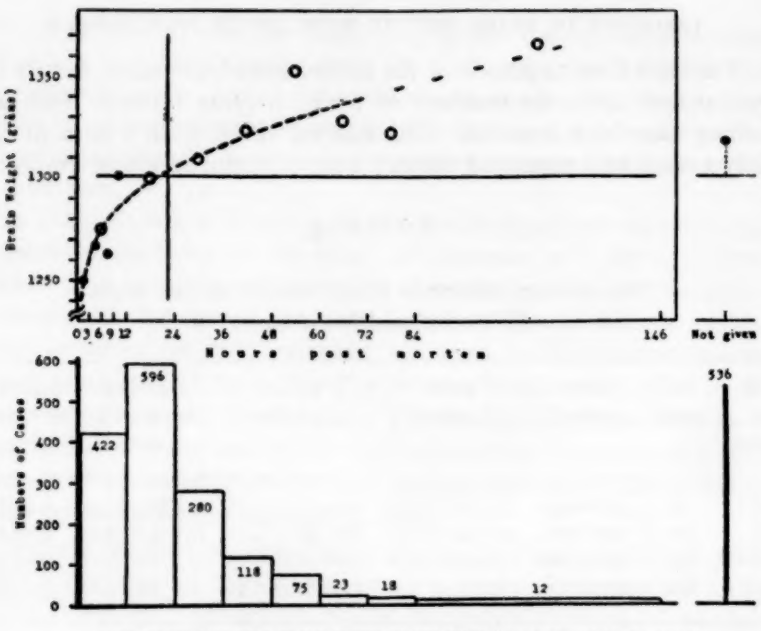
TABLE 4

Post-mortem variation in the weight of the brain
(Non-lesioned-brain cases)

HOURS POST MORTEM	NO. CASES	RANGE *(grams)*	MEAN *(grams)*	MEDIAN *(grams)*	STANDARD DEVIATION *(grams)*	COEFFICIENT OF VARIATION *(percent)*
0–2	23	972–1456	1275.59± 4.12	1270.31± 5.16	125.36± 2.91	9.83±0.23
3–5	141	936–1600	1267.02± 7.00	1259.38± 8.77	123.24± 4.95	9.73±0.39
6–8	127	971–1600	1263.39± 7.28	1260.26± 9.13	121.71± 5.15	9.63±0.41
9–11	131	1020–1729	1301.15± 7.39	1298.61± 9.26	125.34± 5.22	9.63±0.41
0–11	422	936–1729	1275.59± 4.12	1270.31± 5.16	125.36± 2.91	9.83±0.23
12–23	596	950–1720	1300.00± 3.64	1296.84± 4.56	131.80± 2.58	10.14±0.20
24–35	280	1000–1616	1309.64± 4.85	1306.10± 6.08	120.43± 3.43	9.20±0.26
36–47	118	1010–1620	1323.73± 7.07	1320.51± 8.86	113.88± 5.00	8.61±0.38
48–59	75	1070–1600	1352.67± 9.33	1350.00±11.70	119.83± 6.60	8.86±0.40
60–71	23	1120–1600	1328.26±19.00	1293.75±23.82	135.13±13.44	10.18±1.02
72–83	18	1125–1510	1322.22±16.80	1300.00±21.06	105.70±11.88	8.00±0.90
84–146	12	1120–1600	1366.67±25.55	1350.00±32.03	131.23±18.07	9.61±1.33
0–146	1544	.....	1300.65± 2.19	.....	127.53± 1.55	.....
Not given	536	870–1750	1318.66± 3.96	1315.57± 4.96	135.81± 2.80	10.30±0.21
Total	2080		1305.29± 1.92		129.96± 1.36	

hour, 33 within the third; only 44 cases were examined *after* 60 hours, and only 19 *after* 72 hours. The mean number of hours was 22.49.

In the initial study of post-mortem variation the cases were grouped in 12-hour classes; later the first 12-hour class (0-11 hours inclusive) was broken into 3-hour classes. The results of these studies are shown in Table 4 and in Fig. 4.



POST-MORTEM VARIATION IN THE WEIGHT OF THE BRAIN (non-lesioned-brain cases).

Upper graph: The horizontal line indicates the mean brain weight, 1300.65 grams, for the 1544 cases for which the numbers of hours post mortem are given. The vertical line indicates the mean number of hours post mortem, 22.53. Points are located on the field for the mean weights not only for the 12-hour intervals (circles) but also for the first four 3-hour intervals (dots); and off the graph, to the right, is a point for the mean weight of the cases of unrecorded autopsy time. For an explanation of the trend curve (broken line) see the text.

Lower graph: The numbers of cases are shown for each 12-hour interval, and for the long terminal interval.

FIG. 4

The mean and the median weights seem to increase with the number of hours post mortem, the increase being most marked in the early hours. The increase is not great, but from the lowest mean weight to the highest it amounts to 116.67 grams, or 9 percent of the mean weight for the total array. The relative amount of this increase may be compared with the relative amount of the decrease that occurs with age (11 percent).

An increment of 9 percent is biologically very considerable, and even statistically it must be regarded as significant. None of the post-mortem class means differ from the mean for the total array by more than 4 times the probable error of the difference, and only one of the class means, that for the 48—59-hour period, differes from the mean for the first 3-hour period by more than 6 times the probable error of the difference.

But we do not know what the maximal increment may be, and we have no reason to assume that it stops at the limit set by our data. We would like to ascertain the regression of brain weight on hours post mortem. For the data as grouped by 12-hour periods, the coefficient of correlation between brain weight and time post mortem is $+.151 \pm .017$, and the correlation ratio is .164. The squares of these indices differ by only $.0039 \pm .0021$. It should not be concluded, however, that the regression of brain weight on post-mortem time is linear, for on this point the indices as calculated from the grouped data are misleading. The brain weight increases most rapidly during the first 12 hours after death. When the correlation is estimated from the hour-by-hour data, the coefficient is $+.156 \pm .017$, the ratio is .289, and the difference between the squares of these indices is $.0813 \pm .0090$. This difference is more than 9 times its probable error, and it is certainly significant within the limits of error set by the sampling fluctuations. It must be concluded that the regression deviates appreciably from being linear, as indeed it appears to do.

In order to approximate the trend of the data both the hour-by-hour means and the Running means for 5-hour groupings were calculated. The Running means, when graphed, suggested a curve of the form,

$$y = a + bx + c \log x$$

Curves of this form were accordingly fitted not only to the Running means but also to the means for the 12-hour post-mortem periods and the means for the hour-by-hour data. The curve chosen for presentation in this paper is one calculated from the mean weights for the ungrouped data for the first 42 hours after death. This one was chosen because it gave a satisfactory fit not only to the ungrouped data for the first 42 hours but because it fitted the data as originally studied by 12-hour periods. For the first four 12-hour periods the curve fits very closely to the observed means (see figure), and this is what we want, inasmuch as these periods are represented by the most adequate numbers of cases. As a matter of fact the curve lies about as it should for all of the observed means. This curve is not proposed as being an expression of the true regression of brain weight on time post mortem, however, because we do not think we are in a position as yet to determine that regression. It is probable that the post-mortem increase varies both in amount and in rate depending on conditions which will be discussed later. If so, this curve has no precise significance. At best it expresses the mean

increase in this particular array of cases; if so, it indicates the general character of the post-mortem increase, and that, for the purpose of this survey, is sufficient. The equation for the curve is,

$$\text{Brain weight (in grams)} = 1242.44 + .33\,H + 39.72 \log H$$

Here H is the number of hours post mortem.

The curve is certainly suggestive. It suggests that the weight of the brain increases almost continuously after death, at a rate that gradually diminishes. The increase must be limited, but its limit must be beyond the reach of our data. The weight certainly increases for many hours, and possibly it does so for more than 6 days. In the first 12 hours after death the mean weight increases about 47 grams, or nearly 4 percent. By the end of the second 12-hour period the increase amounts to 63 grams, or more than 5 percent; by 36 hours it amounts to 74 grams, or nearly 6 percent. *If* this increase continued for 170 hours after death it would

TABLE 5

Variation in age-at-death by hours post mortem
(Non-lesioned-brain cases)

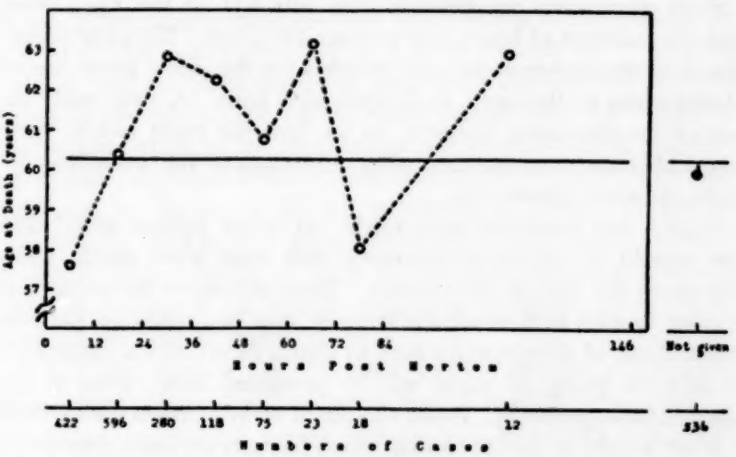
TIME POST MORTEM *(hours)*	NO. CASES	RANGE *(years)*	MEAN *(years)*	MEDIAN *(years)*	STANDARD DEVIATION *(years)*	COEFFICIENT OF VARIATION *(percent)*
0–11	422	18–94	57.62±0.50	58.21±0.63	15.28±0.35	26.52±0.66
12–23	595	19–89	60.41±0.42	62.50±0.52	15.01±0.29	24.85±0.52
24–35	280	17–96	62.84±0.67	66.67±0.84	16.54±0.47	26.32±0.80
36–47	118	24–87	62.25±0.94	66.67±1.18	15.15±0.67	24.34±1.13
48–59	75	26–87	60.77±1.21	65.83±1.51	15.48±0.85	25.47±1.49
60–71	23	29–81	63.15±1.88	66.88±2.36	13.38±1.33	21.19±2.20
72–83	18	31–84	58.06±2.50	57.50±3.14	15.74±1.77	27.10±3.26
84–146	12	42–84	62.92±2.57	62.50±3.22	13.20±1.82	20.98±3.01
0–146	1543	17–96	60.28±0.27	62.81±0.33	15.50±0.19	25.72±0.33
H.P.M. not given	536	12–92	59.69±0.48	62.24±0.61	16.59±0.34	27.80±0.62
Age not given	1					
Total	2080					

amount to 145 grams, or the amount of the mean loss in weight between ages 25 and 96.

Conjectures as to the implications of this increase are not in order until it has been demonstrated beyond any doubt that the increase is a real phenomenon. Other workers have demonstrated that the weight of the brain varies with the skull dimensions and with the age at death, so there has been no reason to doubt our findings on these points. But no evidence of a post-mortem increase has ever before been presented. It is conceivable that the apparent increase might be due to a chance drift in the age at death, or in the dimensions of the skull, though it is difficult to see why the apparent increase should be essentially continuous and at a gradually diminishing rate if any accident of sampling or distribution is responsible for it. Still, the possibility needs to be investigated.

The age distribution is shown in Table 5 and in Fig. 5.

The mean weights for the successive post-mortem periods can be adjusted for age; that is, they can be reduced to the weights they would be if the groups of cases from which they are derived were of the same mean age. In Table 6 the weights have been adjusted to the age-base of 60.28 years, this being the mean age of the 1543 cases.



VARIATION IN AGE-AT-DEATH BY HOURS POST MORTEM, 1544 non-lesioned-brain cases.

The mean age at death, 60.28 years, is indicated by the horizontal line. The numbers of cases are indicated below the graph for each 12-hour period. At the extreme right is indicated the mean age at death of the 536 cases for which the autopsy time was not recorded.

FIG. 5

TABLE 6

Weights adjusted to age-base of 60.28 years

HOURS POST MORTEM	DEVIATION OF MEAN AGE FROM 60.28 YEARS	DEVIATION TIMES THE AGE FACTOR (—2.041)	OBSERVED MEAN WEIGHT *(grams)*	ADJUSTED MEAN WEIGHT *(grams)*
0–11	—2.66	+5.43	1275.59	1281.02
12–23	+ .13	— .27	1300.00	1299.73
24–35	+2.56	—5.22	1309.64	1304.42
36–47	+1.97	—4.02	1323.73	1319.71
48–59	+ .49	—1.00	1352.67	1351.67
60–71	+2.87	—5.86	1328.26	1322.40
72–83	—2.22	+4.53	1322.22	1326.75
84–146	+2.64	—5.39	1366.67	1361.28

The post-mortem increase is still apparent after this relatively insignificant adjustment is made. It could still be described by a curve similar to that which was fitted to the unadjusted data.

Skull dimensions are available for only 631 of the 1544 cases for which the numbers of hours post mortem are given. No adjustments can be made in the post-mortem mean weights for the whole array, therefore, to bring them to the same skull-dimension base. A new study can be made of the 631 cases, however, to see how the brain weight varies in them with time post mortem, after adjustments for variations in age and in the skull dimensions.

In part this study has been made. It offers further proof that the mean weight of the brain increases with time after death, and new evidence on the rate of the increase. Some allowance needs to be made for other factors with which the increase may be associated, particularly the conditions of disease at the time of death, therefore the evidence from this selected group of cases will be presented later, when it can be evaluated more precisely. In the shortened array of cases the variation of the brain weight is similar to that which has already been described; the post-mortem variation could be represented by a curve like that which was fitted to the data for the longer array.

Several attempts have been made to get at the post-mortem changes in other ways. The findings of previous workers have suggested that some degree of statistical association can be found between the weight

of the brain and the weight of the body (see Pearl, 1905). It may be doubted that the relation is a very constant one, but the existence of such a relation suggests that such relations might also be found between various parts of the body and the weight of the brain. In view of the absence from the present statistics of any data on the weight or size of the body as a whole, it would be useful to find some bodily organ the weight or size of which remains unchanged after death, and which at death is proportional in weight or size to the weight of the brain. It should then be possible to group the cases according to the weight or size of this organ, and see whether the weight of the brain then remained proportional to the number of hours post mortem. With this end in view, and for purposes of exploration only, we proceeded to see how the weights of several organs vary with hours post mortem. No constancy was found in the weights of any of these organs after death. The weights of the heart, the kidneys, and the spleen vary even more than the weight of the brain. Apparently the weights of the heart and of the kidneys do not increase or decrease after death in any simple, progressive way; at any rate, no such changes are discernible without detailed analysis. The weight of the spleen drops off sharply and continuously after death, as it would if a quantity of fluid was progressively drained away from it. The weight of the liver changes after death almost as much as the weight of the brain, but in an apparently random way which has no meaning for us as yet. So far no attempt has been made to study the post-mortem changes in the weights of any of these organs with due regard to the many factors which might obscure the post-mortem trends. Eventually such studies may be undertaken, but they cannot be handled properly as subsidiaries to the study of the post-mortem changes in the weight of the brain. Insofar as it went, our survey of the weight variations in the organs named threw no light on the post-mortem changes in the weight of the brain. It merely suggested additional problems of the same kind for further investigation.

It had been noticed that the brain weights tend to be aggregated at certain round numbers. Thus there are 52 cases at 1150 grams, 81 at 1200 grams, 93 at 1300 grams, and so on. The exact distribution of the weights is such that a number of widely separated, sizeable groups of cases, in which the weights are within ± 10 grams of a central value, can be picked out. Six of these groups together include 44 percent of the non-lesioned-brain cases. It was thought that these groups could be used to disprove or to verify our previous findings about the

post-mortem variation of the brain weight, hence the mean numbers of hours post mortem and the mean ages at death were calculated for each of these groups. The mean number of hours post mortem was found to increase as the brain weight increased, and the mean age at death was found to decrease; to this extent our previous findings were verified. But the differences between the highest and the lowest class means (7.96 hours, and 9.69 years) were so much smaller than the standard deviations for the total array of 920 cases (15.73 hours, and 15.72 years) that they could not be taken to mean very much although they were significant (6 times their probable errors). The over-all change in weight was about 200 grams for the six groups of cases, but neither the change in time of 7.96 hours post mortem nor the change in age of 9.69 years nor both together could account for such a great change in weight. This approach to the problem of post-mortem variation in the brain weight has no crucial value, hence no precise data will be presented.

The year of death is known for each of the 2080 non-lesioned-brain cases. It was hoped that this information could be used to check our previous findings. There are secular variations in the brain weight, in the numbers of hours post mortem, and in the age at death. The weight increases slightly but rather indecisively over the period covered by the records. The number of hours post mortem shows a decided secular increase. The age at death, on the other hand, shows no relation to the brain-weight trend. We tried the effect of adjusting the brain weights for age differentials. Adjustments could also be made for variations in the skull dimensions. But when all possible adjustments have been made, we do not believe a well-defined secular increase in the brain weight could be demonstrated, and if it could we do not think it should be ascribed to the secular increase in the number of hours post mortem. This is again a sterile approach to the problem of the post-mortem variation in the brain weight, hence no data on secular trends will be given.

Another proof of the post-mortem increase in the weight of the brain appeared when the variation of the brain weight was studied by causes of death. All discussion of the implications of the post-mortem increase will be postponed, therefore, to allow the presentation of the findings described in the next section.

(To be concluded)

A STUDY OF PHYSIOLOGICAL FACTORS: THE AUTONOMIC NERVOUS SYSTEM AND THE SKELETAL MUSCULATURE

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THIS study constitutes the first collation of a number of physiological data from the one hundred and seventy children being studied by the Fels research staff. Since children of school age are more amenable to laboratory work than are younger subjects and hence afford more data, this older group, which at the time of measurement numbered 62, was selected for study. These 62 children ranged in age from 6 to 11 years. Girls and boys were almost equally represented.

Although most of these children have been studied since the fetal period and some information of a physiological nature has been gathered from the inception of the study, a group of new measurements was added to the examination schedule in the summer of 1939. Specifically, these new measures were directed toward the evaluation of physiological status with particular regard for the functioning of the skeletal musculature and of the autonomic nervous system. The larger purpose of the work is to test the postulate that there are significant relationships between physiological functions and personality.

These postulated relationships have furnished the theme for much research in past years. In general, physiological and psychological variables have been compared, two at a time, by routine correlation or by less exact techniques. Almost without exception, coefficients of correlation have been low but suggestive. Results of research in this field might be summarized as being indecisive, but of an order to goad experimenters to further investigation.

Methods are available now, however, for a more exhaustive attack upon these psychophysiological problems. The advent of factor analysis already has furnished new and parsimonious views of mental activity.

Statistically described factors of personality and physique are being charted with great zeal. Possibly it is time to explore statistically the physiological functions of individuals. If valid factors can be discovered in this area, new avenues will be opened for the description of psychophysiological relationships. Although correlations between individual physiological and psychological variables have been low, correlations between physiological and psychological factors may prove significant.

At least three physiological systems give considerable promise of factors which may be not only quantitatively described but also psychologically significant. These are the autonomic nervous system, the skeletal muscular system, and the endocrine system. Many years ago Eppinger and Hess (5) conceived of a sort of continuum of individual differences in functioning of the autonomic nervous system. At one extreme were those in whom the thoraco-lumbar branch supposedly was predominant. They were called the "sympathicotonics." At the other extreme were those in whom the cranio-sacral branch (or at least the vagus nerve) was believed predominant. They were called the "vagotonics" or occasionally the "parasympathicotonics." Although many workers have considered the validity of this hypothesis, none has proved it—none has disproved it. Most have believed that it was adequate for describing a few but not many individuals. Only one worker, Darling (1), has attacked the problem by means of the factorial methods.

Another possible physiological factor with psychological significance is tension of skeletal musculature. The case for such a factor, with some preliminary experimental results, has been presented in an earlier report (13). It was claimed there that the factor methods might make possible the indirect measurement of this phenomenon which is extremely difficult to appraise directly.

A third area of particular interest is the endocrine system and its various ramifications. The many postulated relations between the ductless glands and physique, intellect, personal-social behavior, and the autonomic nervous system itself, seem to invite analysis by the factorial methods.

The present report, however, involves only one obvious measure of endocrine variability—basal metabolic rate. Others are or will be available in the Fels study as our subjects reach adolescence. Immediate efforts have been directed primarily toward measurement of the

autonomic nervous system, with the measurement of muscle tension a not less important secondary interest.

The general scheme of the present work is to measure as many as possible of the functions of the body which are innervated by both the sympathetic and the parasympathetic systems. Then, if these systems are operating more than less antagonistically, and if each is operating even partially as a unit, we should find correlated distributions for some of these functions, and have at hand the tests which should define the postulated autonomic factor and make possible its measurement. An assumption which underlies the work is that physiological status is not essentially different from physiological reactivity. Hence individual differences in status are presumed to reflect individual differences in the underlying stimulation-response complex.

THE NATURE OF THE TESTS

In the selection of the tests it has been necessary to consider the longitudinal nature of the Fels study and the problem of maintaining the cooperation of both children and parents. Drugs, therefore, were eliminated from consideration. What remained of tests suggested in the literature was little enough. Of the functions clearly influenced by both systems, salivary output and heart rate seemed the easiest to determine in our subjects. The latter was recorded electrically with the subject reclining. Rather than attempt the use of the Lashley disc for measuring salivation, a more simple technique was devised. The child was asked to bring as much saliva to the front of his month as he could. This forced output was withdrawn by a suction pump and accumulated before him for a five minute period in a graduated centrifuge tube. The procedure is novel to the subject and furnishes optimal motivation. All salivary tests were made between 10:30 a.m. and 12:00 noon. Since varying amounts of sediment were noticed in the salivary samples, and since Kuntz (8, p. 287) has mentioned that the per cent of total solids in saliva of dogs was increased after electrical stimulation of the sympathetic nerves supplying the salivary glands, the salivary samples were centrifuged and the per cent of total solids recorded.

The electrocardiograms afforded another measure which has been mentioned as being symptomatic of vagotonia; that is, arrhythmic beating of the heart. As reported elsewhere (11), sinus arrhythmia is present in varying degree in most of our subjects and is negatively

related to heart rate. Here then is a fourth test which should assist in determining the axis of the postulated factor.

Of the other 16 variables included in this analysis all (even basal metabolic rate) have been cited at one time or another as bearing some relationship to autonomic function. Skin conductance according to Darrow (3) is closely related to sweating, and certainly both emotional and heat regulatory sweating are mediated by the autonomic system, though in rather a complex manner. All conductance measurements were made with Darrow's circuit (2) and with zinc-cup electrodes 2 cm. in diameter filled with a 1 per cent zinc sulphate agar jelly. For three of the measures (33, 37, and 38) the electrodes were attached to the palms. Variable 39, non-palmar conductance, was recorded from identical electrodes on the wrists. Variable 33 represents the difference between standing and reclining palmar conductance translated to logarithms in order to equate measurements fluctuating around different means (4). A positive change in resistance or a negative change in conductance indicates relaxation of the skeletal musculature (6, 12). Here, then, is the first variable to determine the postulated factor of muscular tension.

Work by Lewis (9) and by Patek and Weiss (10) suggested that dermographia might add something to the picture. Presumably, vagotonia should be characterized by varying degrees of relative dilation in the peripheral blood vessels. In the relative absence of innervation for constriction, a local reflex vasodilatation produced by a scratch (that is, red dermographia) might be expected to persist longer than if innervation for vaso-constriction were predominant. It seemed possible that the latency of this vaso-motor response also might be a function of the amount of vaso-constriction present. These two measures, 22 and 23, were obtained by scratching the skin of the right upper arm with an adjustable stimulator similar to that employed by Patek and Weiss, calibrated at 250 grams pressure. By means of a stopwatch the latency of the red response in seconds and its persistance in minutes were determined. The subjects were reclining for all measures.

The influence of sympathetic stimulation upon respiration rate and systolic blood pressure is well known. Variable 75, the difference between standing and reclining systolic pressure, is the so-called orthostatic hypotension test which has been regarded as significant for distinguishing between vagotonia and sympathicotonia. The

preliminary factor analysis previously mentioned (13) suggested that diastolic and pulse pressure might be related to a muscular factor. This same analysis also offered some suggestion of the significance of variability of respiration amplitude as a measure of muscular tension. This latter variable, 45, was measured in terms of the standard deviation, from the basal metabolism charts. Respiration rate was recorded polygraphically at the same experimental session with the heart, blood pressure, skin conductance, and dermographic measures. All were determined under conditions of relatively constant temperature and humidity, and in the morning hours with the subjects in a resting state.

Although many other tests have been tried experimentally, most have been discarded as valueless or unreliable. Only three have been included in this analysis. They are 32, simple motor reaction time to visual stimulation; 35, ratings of restlessness in the schoolroom as made by the children's teachers; and 34, residual muscle tension of a segment (in this case, the right arm), as measured by Kreezer's (7) technique. The forearm is supported at the wrist in a freely swinging horizontal position and the upper arm secured. After the subject is instructed to relax, passive resistance to mechanical abduction of the forearm is recorded polygraphically. A rapid and smooth curve of abduction indicates a minimum of residual tension, while a slow and/or jerking movement indicates relatively more residual tension. Measurement is in terms of the angle of the curve of abduction.

For the majority of the above measures the data finally employed consisted of means derived from a series of measures on the same day. The salivary and dermographia measurements, the BMR's, and the ratings of restlessness are exceptions to the above, since each is the result of one test. All tests, excepting possibly the ratings of restlessness and the measures of change in systolic blood pressure, are regarded as reliable for the day on which they were recorded. When the mean of a series of data was employed a sufficient number of measures was made to insure a reliable datum. Experimentation has shown repeatedly that the salivary and dermographic results can be duplicated with but slight variations. Any BMR's which were not regarded as satisfactory were repeated on another day.[1]

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[1] The writer is indebted to Virginia Torbet of the Fels staff for the use of BMR data obtained by her.

The question of stability of physiological measures is the next to arise. Since physiological responses are notably quite variable, will measures of physiological status for any single day have much predictive value? At the initiation of these measures during the summer and fall of 1939, repeated tests for a small number of cases over a period of two weeks indicated that at least for that short time most of the functions remained relatively stable. The full battery of tests was given for the first time to all 62 children during the months of January to March, 1940. It is this 1940w body of data which is analyzed in the present report. Most of the tests were repeated during the summer of 1940 and again during January to March 1941. Correlations between the

TABLE 1

The stability of the measures over periods of six and twelve months

		CORRELATIONS BETWEEN DATA	
		1940w and 1940s $n = 45$	1940w and 1941w $n = 60$
22	Dermographia latency	.33	.03
23	Dermographia persistence	.39	.43
25	Salivary output	.70	.69
26	Percent solids in saliva	.33	..
28	Heart period	.64	.67
29	Sinus arrhythmia	..	.71
32	Reaction time	.57	..
33	Change in palmar log conductance	.37	.11
34	Muscular relaxation	(.16)*	..
35	Restlessness (rating)	Retest data not yet available	
37	Standing palmar conductance	.23	.40
38	Reclining palmar conductance	.29	.35
39	Non-palmar conductance	—.13	.37
43	Basal metabolic rate	Retest data not yet available	
45	Sigma respiration amplitude	Retest data not yet available	
58	Respiration rate	.34	.40
75	Change in systolic blood pressure	..	.38**
76	Systolic blood pressure	.69	.70
77	Diastolic blood pressure	.49	.60
80	Pulse pressure	.36	.39

* (1940w and 1939s, $n = 34$)

** ($n = 43$)

TABLE 2 *Intercorrelations between twenty physiological variables for 62 children, age six to eleven years, age partialled out*

TABLE 2 *Intercorrelations between twenty physiological variables for 62 children, age six to eleven years, age partialled out*

	22	23	25	26	28	29	32	33	34	35	37	38	39	43	45	58	75	76	77	80	AGE
22 Derm. latency	...	—.12	—.05	.03	.00	.29	—.26	—.08	—.05	—.10	—.04	.01	.19	—.03	—.05	—.19	.02	—.12	—.28	.19	—.28
23 Derm. persistence	...	...	.10	—.08	—.04	.16	.27	.24	.08	—.07	—.06	—.15	.09	.03	—.08	—.16	.01	.05	.03	.00	—.18
25 Salivary output	...	...	...	—.48	.19	.25	.03	—.07	.07	.00	—.14	—.09	—.18	—.14	.07	—.02	—.03	.21	—.02	.13	.16
26 Percent solids in salvia	...	...	...	...	—.15	—.35	.10	.08	.04	—.17	.19	.09	—.01	.11	—.04	.08	—.12	—.13	.13	—.22	.17
28 Heart period	...	...	...	...	...	.68	—.24	—.30	—.15	.00	—.04	.09	—.03	—.40	.18	—.32	—.02	—.21	—.39	.22	.30
29 Sinus arrhythmia	...	...	...	...	...	...	—.28	—.22	—.22	.02	—.06	.04	.00	—.16	—.05	—.25	—.10	—.03	—.20	.18	.03
32 Reaction time	...	...	...	...	...	...	...	.24	.25	.07	—.03	—.10	.04	—.12	—.07	—.02	—.04	—.13	—.09	—.13	—.45
33 Change in palmar log conductance	...	...	...	...	...	...	...	...	.29	—.34	.06	—.51	—.04	.25	—.05	.07	.03	.08	.24	—.16	—.01
34 Muscular relaxation	...	...	...	...	...	...	...	...	...	—.07	.09	—.07	—.08	—.04	.05	.05	—.14	—.15	—.17	—.04	.10
35 Restlessness (rating)	...	...	...	...	...	...	...	...	...	...	—.02	.20	—.03	—.12	—.05	.19	.03	—.08	—.13	.03	—.12
37 Standing palmar conductance	...	...	...	...	...	...	...	...	...	...	...	.80	.27	.06	—.10	.09	—.01	—.02	—.01	—.01	—.12
38 Reclining Palmar conductance	...	...	...	...	...	...	...	...	...	...	...	...	.26	—.04	—.08	.04	—.09	—.07	—.12	.06	—.14
39 Non-palmar conductance	...	...	...	...	...	...	...	...	...	...	...	...	...	.05	—.10	—.09	—.07	—.08	—.02	—.07	—.08
43 Basal metabolic rate	...	...	...	...	...	...	...	...	...	...	...	...	...	...	.09	.08	—.28	.02	.23	—.17	.03
45 Sigma respiration amplitude	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	—.45	.03	—.10	.14	—.31	—.04
58 Respiration rate	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	.05	.07	.10	.03	—.18
75 Change in systolic blood pressure	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	.03	.12	—.06	—.16
76 Systolic blood pressure	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	.46	.39	.49
77 Diastolic blood pressure	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	— 56	.07
80 Pulse pressure	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	.34

1940w and 1940s data and between the 1940w and 1941w data are shown in Table 1.

It will be seen that in general those from winter to winter are higher than those from winter to summer. Evidence to be reported elsewhere (14) indicates that many of these physiological responses differ from winter to summer and that this variation is not in the same direction for all individuals. Relationships from winter to winter are interpreted, therefore, as being the more valid. For their evaluation it should be kept in mind that insofar as a given coefficient is positive it reflects not only reliability of measurement but stability of response. If it is low or negative it indicates inconsistency of measurement, but not necessarily unreliability of measurement. It should be noted also that these children have been subjects of the Fels study from six to eleven years and are quite accustomed to such examinations and to the personnel making the tests. The present coefficients, therefore, probably are somewhat higher than might be expected in an uninitiated child population.

RESULTS

The intercorrelations of these twenty variables with chronological age partialled out are shown in Table 2. The correlations with age are shown in the last column of the table. It will be noted that in general the correlations shown in the table are very low. Their mean magnitude and σ is .13. It will also be seen that the table is replete with negative correlations. These could not be eliminated entirely by reflection, so it was impossible to follow up the earlier work (13) with an analysis by Holzinger's bi-factor method. The centroid method of Thurstone yielded the pattern shown in Table 3. Positive coefficients were maximized by reflection of the seven variables marked with an X.

The first question which must be considered is — how many factors are represented? When should factoring be stopped? Judging by the size of the residuals the data suggested five factors, but the last three were indicated largely by sizable residual correlations between tests of specific function.

Factor K_3 was largely attributable to the high correlation between tests 37 and 38, standing and reclining palmar conductance. Variable 38 was recorded only to make possible measure 33, the change in log conductance between the standing and reclining positions, which is derived from both 37 and 38. Had 38 been omitted from the analysis it

TABLE 3

The Centroid Factor Pattern of Data from Table 2

		VARIABLES (X DENOTES REFLECTED TRAIT)	K_1	K_2	K_3	K_4	K_5
22	X	Short latency dermographia...	.26	.19	.01	.18	—.27
23		Long persistence dermographia	.17	.30	.34	—.16	.09
25	X	Low salivary output.....	.28	—.48	—.21	—.17	.22
26		High percent solids in saliva	.37	—.39	—.11	—.10	.18
28	X	Fast heart rate.....	.74	—.12	—.15	.22	.06
29	X	Little sinus arrhythmia.....	.65	—.20	—.16	.28	.12
32		Long reaction time.....	.29	.11	.15	.18	.35
33		Large change palmar log. cond.	.64	.37	.24	—.29	.12
34		Low residual muscle tension...	.17	.06	.22	.17	.30
35	X	Quiet, not restless (rating)....	.20	.07	.19	—.34	.07
37		High std. palmar conductance	.18	—.45	.50	.29	—.27
38		High rec. palmar conductance	—.17	—.16	.34	.47	—.35
39		High non-palmar conductance	.05	—.31	.18	—.07	.09
43		High basal metabolic rate.....	.30	—.25	.10	—.13	—.06
45	X	Low sigma respiration ampl....	.14	.15	—.07	.33	.17
58		Fast respiration rate.....	.30	.06	—.33	.49	—.12
75		Large change systolic B. P.....	.01	.13	—.30	—.02	—.09
76		High systolic blood pressure...	.11	.37	.02	.15	—.29
77		High diastolic blood pressure	.53	.04	—.13	—.22	—.65
80	X	Low pulse pressure.....	.35	—.22	—.16	—.42	—.29
		Mean of factor residuals	.10	.10	.09	.08	.07
		Sigma of factor residuals	.10	.08	.08	.07	.06

seems probable that Factor K_3 would have been quite different. Factor K_4 seemed to be indicated largely by the high residual intercorrelation of heart period, 28, and sinus arrhythmia, 29. Factor K_5 seemed to be suggested by the high residual correlation of 77, diastolic blood pressure, and 80, pulse pressure, which is derived from variables 76 and 77.

All five factors were used in the first rotated solution. The results after seven rotations are shown in Table 4. It is believed the first factor may be interpreted as representing the autonomic nervous system, although the interpretation is by no means decisive. The salivary measures are most highly correlated with it and only the low but logical relationships with dermographia persistence, heart rate and sinus arrhythmia, non-palmar conductance and basal metabolic rate, call for any other definition than in terms of salivation per se.

TABLE 4. *Three rotated solutions of the centroid pattern*

VARIABLES (X DENOTES REFLECTED TRAIT)	PATTERN FOR FIVE FACTORS						PATTERN FOR THREE FACTORS				PATTERN FOR TWO FACTORS		
	I	II	III	IV	V	h^2	A	B	C	h^2	Nu	Mu	h^2
22 X Short latency dermographia...	—.25	.03	.02	.25	.29	.21	—.04	.32	.00	.10	—.06	.31	.10
23 Long persistence dermographia	—.16	.42	.04	—.20	.15	.27	—.28	.29	.27	.23	—.20	.28	.12
25 X Low salivary output.....	.62	.01	.14	.15	.02	.43	.59	.00	—.01	.35	.56	.05	.32
26 High percent solids in saliva	.50	.09	.20	.19	.08	.34	.53	.12	.07	.30	.52	.17	.30
28 X Fast heart rate.....	.23	.27	.22	.62	.27	.63	.49	.58	.01	.58	.42	.62	.56
29 X Little sinus arrhythmia.....	.25	.21	.27	.61	.15	.58	.52	.46	.01	.48	.46	.51	.47
32 Long reaction time.....	—.01	.44	.18	.19	—.12	.28	.00	.31	.16	.12	.02	.31	.10
33 Large change palmar log cond.	.03	.70	—.01	.02	.46	.71	—.08	.74	.23	.61	—.07	.74	.55
34 Low residual muscle tension	—.05	.35	.22	.08	—.15	.20	—.04	.17	.22	.08	.02	.18	.03
35 X Quiet, not restless (rating)...	.13	.29	.01	—.22	.23	.21	—.03	.21	.19	.09	.02	.21	.04
37 High std. palmar conductance	—.11	—.14	.73	.01	.21	.65	.29	—.07	.63	.49	.48	—.03	.23
38 High rec. palmar conductance	—.13	—.56	.74	.03	—.07	.88	.32	—.46	.47	.54	.50	—.42	.43
39 High non-palmar conductance	.21	—.01	.29	—.11	—.02	.14	.22	—.11	.26	.13	.30	—.08	.10
43 High basal metabolic rate....	.25	.04	.23	.02	.26	.18	.32	.13	.21	.16	.36	.17	.16
45 X Low sigma respiration ampl...	—.14	.18	.02	.33	—.16	.19	—.03	.20	—.05	.05	—.07	.19	.04
58 Fast respiration rate.....	—.14	—.07	.00	.65	.04	.45	.20	.29	—.28	.20	.07	.30	.09
75 Large change systolic B. P....	.00	—.08	—.28	.16	.07	.12	.00	.08	—.32	.11	—.11	.06	.02
76 High systolic blood pressure	—.40	.04	—.11	.16	.24	.25	—.25	.28	—.07	.15	—.29	.26	.15
77 High diastolic blood pressure	.02	—.12	—.07	.20	.84	.78	.27	.48	—.04	.30	.19	.50	.29
80 X Low pulse pressure.....	.39	—.09	—.06	.00	.54	.45	.39	.20	—.03	.19	.35	.23	.18

Factor II may, possibly, be termed "relaxability" — that is, the trait of either *being* relaxed muscularly, or *being able* to relax quickly upon sitting or reclining. It is most highly correlated with the change in palmar log conductance which is claimed to be essentially a measure of relaxation of skeletal musculature. Further, it involves reclining palmar conductance from which the above measure was partially derived, plus the measure of residual tension in the arm, the rating of restlessness, and reaction time, all of which certainly are related to the skeletal musculature. It is of interest that dermographia persistance shows its highest loading on this factor.

Factor III seems to be largely specific to skin conductance. Factor IV seems to be a "rate" factor, weighted as it is with heart and respiration rate and variability in these two functions, the variability of each being in inverse proportion to rate. Factor V can, perhaps, be defined in terms of diastolic blood pressure and pulse pressure. Why variable 33, change in log conductance, has a loading here is a question, unless it indicates a second muscular tension factor.

Looking again at the means and sigmas of the factor residuals, and taking cognizance of the fact already cited that the last three factors had been indicated largely by high residual correlations between test doublets representing the same or similar processes, it seemed possible that the data had been over-factored. A second solution was undertaken, therefore, using only the first three factors of the centroid pattern. The result after two rotations appears in Table 4.

From a theoretical point of view, factor A in this second solution is a more imposing autonomic factor than was factor I in the first solution. Factor B is perhaps not less convincing than factor II as a muscular factor. Factor C again is largely specific to the skin conductance measures. It is of incidental interest that K_2 of the centroid pattern, where no rotations at all were made, is not greatly different from the rotated factors A and I.

It seemed possible that only two significant common factors were represented in these data and that the interrelationships among the skin conductance data were so great that they constituted a specific third factor of sufficient magnitude to confuse the true picture. On this assumption a two factor solution was tried. K_1 and K_2 alone were rotated against each other. The results are shown in Table 4. It is obvious that the assumed orthogonality of these two factors may be questioned. Since neither factor is as yet precisely delineated, however,

the experimenter chooses to be parsimonious and regard them, for the present, as unrelated. Such a position is, of course, little more than hypothetical. It assumes that when the factors are better measured they will be found orthogonal.

In the opinion of the writer, this two-factor solution affords the most meaningful description of the data. The reasons for this opinion are set forth in the following paragraphs.

DISCUSSION

As was stated in the introduction, if the parasympathetic and sympathetic systems are operating in these subjects more than less antagonistically, and if each is operating even partially as a unit, we should expect to find functions mediated at least in part by these systems to be intercorrelated. In other words, if individuals do differ in

TABLE 5

A theoretical picture of autonomic response relationships and the emperical patterns derived from four factor solutions

NO.	TEST	PARASYMPATHETIC PREDOMINANCE	SYMPATHETIC PREDOMINANCE	FACTOR K_3	I	A	Nu
22	Dermographia latency.....	Short	Long	—.19	—.25	—.04	—.06
23	Dermographia persistence	Long	Short	.30	.16	.28	.20
25	Salivary output.....	High	Low	.48	.62	.59	.56
26	Percent solids in saliva.....	Low	High	.39	.50	.53	.52
28	Heart rate.....	Slow	Fast	.12	.23	.49	.42
29	Sinus arrhythmia.....	Much	Little	.20	.25	.52	.46
37	Standing palmar conductance...	Low	High	.45	—.11	.29	.48
39	Non-palmar conductance.....	Low	High	.31	.21	.22	.30
58	Respiration rate.....	Slow	Fast	—.06	—.14	.20	.07
75	Increase in systolic blood pressure	Little or drop	Much	—.13	.00	.00	—.11
76	Systolic blood pressure.....	Low	High	—.37	—.40	—.25	—.29
	Mean of positive correlations with autonomic factor			.32	.33	.39	.38
	Number of variables in predicted direction			7	6	8	8

autonomic function per se, logical relationships should be expected among discrete autonomic functions. In terms of the present battery of supposed best autonomic tests, extreme subjects should present somewhat the picture shown in the first columns of Table 5.

In the last four columns of the same table appear the appropriately reflected correlations of the tests with the supposed autonomic factor from each of the four solutions. If individuals do differ autonomically and if the tests are valid measures of discrete aspects of those differences, all correlations should be positive. Since all four solutions show the majority of the correlations to be positive it follows that there is a certain amount of communality among some of these tests which probably reflects individual differences in autonomic function. It follows also that for these 62 subjects, the hypothesis of Eppinger and Hess is, in some measure, substantiated.

Since factors A and Nu are seen to fit the predictions better than factors K_2 and I, it is assumed that they furnish the more valid descriptions of the autonomic factor. Since factor Nu includes some of the variance of tests 37 and 39 which, in the other solutions, was believed to have been relegated erroneously to a specific factor, and since in other respects it is quite similar to factor A, it is believed to describe this autonomic factor best.

It is not surprising that variables 22 and 75, dermographia latency and change in systolic blood pressure upon rising, do not fall into this logical picture. In recent work, the former continues to show great and apparently unsystematic variability. The latter undoubtedly was influenced greatly by muscular activity, and is being investigated anew with the "tilt-table" technique. It is surprising, however, that 76, systolic blood pressure, should show a negative correlation. Any explanation of such apparent discrepancy must await further analysis.

As far as the muscle tension factor is concerned, factor Mu from the two factor solution is believed to be not less and probably more significant than factor II of the five factor solution. Factor B of the three factor solution does not differ significantly from factor Mu. A partial comparison of the two factorial descriptions, Mu, and Factor II is shown in Table 6, along with seven comparable measures and their correlation with the supposed muscular factor Δ in the earlier analysis (13). While the results are certainly not decisive, there is a distinct similarity between either II or Mu and factor Δ. The latter two bring diastolic blood pressure and pulse pressure into the picture, however,

TABLE 6

The muscular factor from two solutions compared with factor Δ from an earlier solution

NO.		TEST	FACTOR II	FACTOR Mu	FACTOR Δ FROM (13)	TEST NO. FROM (13)
22	X	Short dermographia latency.....	.03	.31	...	...
23		Long dermographia persistence.....	.42	.28	...	...
28	X	Fast heart rate.....	.27	.62	...	...
29	X	Little sinus arrhythmia.....	.21	.51	...	...
32		Long reaction time.....	.44	.31	...	...
33		Large change palmar log conductance	.70	.74	.56	32
34		Low residual muscle tension.....	.35	.18	...	...
35	X	Quiet, not restless.....	.29	.21	.56	Cx
38		High reclining palmar conductance	—.56	—.42	—.64	31x
43		Basal metabolic rate.....	—.09	.17	—.27	29
45	X	Low sigma respiration amplitude....	.18	.19	.17	Gx
77		High diastolic blood pressure.....	—.12	.50	.80	27
80	X	Low pulse pressure.....	—.09	.23	.25	28x

while factor II does not do so. Although factor Δ is not as good a validity criterion as might be desired, it appears to be the only one available as yet. It may be tentatively concluded then that factor Mu of the two factor solution is a more acceptable description of this muscular factor than is factor II of the five factor solution, and that the similarity of factors Mu and Δ lends some credence to the validity of both.

Only after other similar analyses have been completed and after an attempt has been made to validate the factors by means of estimated factor scores for individuals, can it be known whether or not any of these solutions is significant. At the moment Darling's (1) analysis is the only one available for comparison with the autonomic factor. Since the present data afford only three or possibly four variables similar to his, the factor patterns can hardly be expected to have much in common. It is interesting to note, however, that he describes a cholinergic or parasympathetic factor, and an adrenergic or sympathetic factor, instead of the one autonomic factor reported here.

Other analyses will be made in this laboratory as more data are accumulated. At the moment, work with estimated factor scores affords

evidence for regarding factor Nu as valid and fairly consistent in this population over periods of six months and one year. Analysis of the estimates for factor Mu is in progress. This work is to be reported elsewhere (15, 16).

SUMMARY

Twenty physiological variables as measured in a population of 62 school age children (1940 winter data) have been intercorrelated and submitted to factor analysis. Three rotated solutions involving five, three, and two factors respectively have been presented. The two-factor solution is believed to best describe the essential data. The two factors have been defined tentatively as representing the "autonomic nervous system" and "relaxability of skeletal musculature." The autonomic factor is delineated the more clearly, and is believed to offer both substantiation for the hypothesis of Eppinger and Hess and the means of measuring the individual differences in autonomic function which they postulated.

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ON THE VARIANCE OF HUMAN LIVE BIRTH SEX RATIOS

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THE cytological studies of Winiwarter (1912), Painter (1923), Oguma and Kihara (1923), Evans and Swezy (1929), as well as those of others, suggest that human sperms are of two kinds with respect to sex determination and that these two kinds are produced in equal numbers.

If these deductions are correct and if all other factors are assumed constant, one should, of course, expect a human live birth sex ratio of 50 per cent males to 50 per cent females. As is common knowledge this ratio is seldom if ever realized in any population of large size. Generally there are more males than females born. For instance, there were 1,088,387 live births reported to the Chicago Board of Health for the Chicago Area from 1919 to 1938 inclusive. Of these, 559,906 or 51.444 per cent were males and 528,481 or 48.556 per cent were females. This is an excess of 1.444 per cent males.

That the above indicated excess is a significant one can be readily shown:

$$\text{S.E.} = \sqrt{\frac{p \cdot q}{n}} = \sqrt{\frac{.50 \times .50}{1{,}088{,}387}} = .000479$$

$$\frac{\text{Difference}}{\text{S.E.}} = \frac{.01444}{.000479} = 30.1.$$

Since the difference of two times the standard error is generally considered significant, there can be no question but that the above indicated sex ratio is a significant deviation from the expected 50:50 ratio. The same could be shown for the live birth sex ratio of most other large populations.

If live birth sex ratios deviate significantly from a 50:50 ratio, the question arises as to the cause or causes of these deviations. The explanation which first comes to mind probably is that more female than male

embryos die in utero. While this is a possible explanation it is not supported by facts. There are actually more male than female stillbirths at every period of gestation. In fact the preponderance of male stillbirths is so great that the fertilization or conception ratio may be as high as 52 per cent males to 48 per cent females or even higher. In other words the live birth sex ratio is a function of at least two sets of factors (1) factors that tend to bring about the fertilization of more eggs by male than by female determining sperms, and (2) factors that tend to result in the death of more males than females in utero and at time of birth.

As an explanation of the greater percentage of males conceived it has been suggested that the male determining sperms are more mobile than the female determining sperms and therefore more of them reach the upper ends of the oviducts in time to fertilize the eggs. Another possible explanation, although not a very probable one, is that more female than male determining sperms are killed by lethal genetic factors and hence a higher percentage of the latter reaches the eggs. A third possible explanation is that the female determining sperms, for reasons other than the ones already indicated, have a lower survival value in the chemical environment of the female genital tract than do the male determining sperms and hence fewer reach the upper ends of the oviducts. While all these explanations are probable ones, there is no direct proof that any one of them is correct. The greater number of male than female prenatal deaths has been attributed to sex-linked lethal genes and to secondary sex differences which give a differential survival value in favor of the females. But again no clear demonstration of the correctness of either of these two hypotheses has been given.

Though it may not be possible for some time to analyze in detail the factors responsible for the significant deviation of the live birth sex ratios from a 50:50 ratio, it is possible to learn something about their constancy or variability as a unit over a period of time or under different environmental and genetic conditions. This can be done in various ways. It is possible to obtain the mean live birth sex ratio for different periods of time and for different sets of environmental and genetic conditions and then test the differences between the means for significance. This has been done by a number of investigators for various populations and their studies have contributed much valuable information. Some of this information will be referred to later.

TABLE 1

*Live births for the Chicago Area from 1919-1938 as reported to the Chicago Board of Health**

YEAR	NO. OF BIRTHS	NO. OF ♂	PERCENT ♂
1919	46,758	24,103	51.548
1920	55,316	28,293	51.148
1921	62,611	32,212	51.448
1922	56,724	29,170	51.424
1923	55,934	28,905	51.677
1924	58,900	30,196	51.267
1925	59,639	30,779	51.609
1926	60,200	30,772	51.116
1927	60,888	31,331	51.457
1928	59,016	30,337	51.405
1929	58,800	30,210	51.378
1930	58,083	29,881	51.445
1931	52,993	27,464	51.826
1932	49,258	25,310	51.382
1933	46,655	24,004	51.450
1934	47,955	24,734	51.578
1935	49,425	25,392	51.375
1936	47,939	24,698	51.520
1937	49,633	25,563	51.504
1938	51,660	26,552	51.398
Total	1,088,387	559,906	
Mean	54,419.35	27,995.3	51.444

$$\sigma^2_0 = \frac{fd^2}{n-1} = \frac{.0000506135}{19} = .00000266$$

$$\sigma^2_T = \frac{.51444 \times .48556}{54{,}419.35} = \frac{.2497914864}{54{,}419.35} = .00000459$$

$$F = \frac{\sigma^2_T}{\sigma^2_0} = \frac{.00000459}{.00000266} = 1.72 = \text{Probability greater than } .05.$$

* The writer is indebted to Dr. Herman N. Bundesen, President of the Chicago Board of Health, for supplying the live birth data for the Chicago Area and for permission to use it in this publication.

Another way of testing for the constancy of the factors responsible for the deviation in live birth sex ratio from a 50:50 ratio is to compare the variance of observed sex ratios of different populations with the variance expected on the basis of chance. This paper concerns itself primarily with the application of this method to the sex ratios of a number of different populations.

In Table 1 are given the yearly percentages of male live births in the Chicago Area from 1919 to 1938 inclusive. The mean percentage for this period is 51.444. As shown above this percentage is a significant deviation from the expected 50 per cent. The variance of these percentages is:

$$\sigma^2_0 = \frac{\Sigma f d^2}{n-1} = \frac{.0000506135}{19} = .00000266.$$

The theoretical variance or the variance expected on the basis of chance is:

$$\sigma^2_T = \frac{p \cdot q}{n} = \frac{.51444 \times .48556}{54419.35} = .00000459.$$

Comparing the two variances we find that the observed variance is not greater than that expected due to random fluctuation. In fact the theoretical variance is 1.72 times greater than the observed. This deviation raises the question of whether the observed variance is significantly smaller than that expected due to random fluctuation. R. A. Fisher (1936) has calculated the chance distribution of F (the relation of Variance$_1$ to Variance$_2$) for different degrees of freedom. Snedecor (1937) has prepared tables which enable one to obtain directly the probabilities of values of F. In looking up the probability of $F = 1.723$ we find that it has a probability greater than .05. In other words, the variance of the live birth sex ratio for the Chicago Area from 1919 to 1938 does not deviate significantly from that expected due to chance. This means that the factors which are responsible for the deviation of human live birth sex ratios from a 50:50 ratio have as a unit not varied in the Chicago Area over a period of twenty years more than might be expected due to random fluctuations. This result is in accord with the general findings of Ciocco (1938) who compared the means of sex ratios of different populations and found that they did not differ significantly under fairly different external environmental conditions. This result also is in agreement with Ciocco's conclusion that "the sex ratio

TABLE 2

Live births for the forty-eight states and the District of Columbia of the U. S. Birth Registration Area for the year 1935

STATES	NO. OF BIRTHS	NO. OF ♂	PERCENT ♂
Ala.	62,239	31,825	51.134
Ari.	9,139	4,655	50.936
Ark.	35,684	18,457	51.723
Calif.	80,131	41,166	51.373
Col.	18,837	9,651	51.234
Conn.	22,258	11,398	51.209
Del.	4,036	2,079	51.511
D. C.	10,803	5,568	51.541
Fla.	28,051	14,403	51.346
Ga.	63,260	32,299	51.058
Idaho	9,469	4,838	51.093
Ill.	111,884	57,294	51.208
Ind.	52,909	27,214	51.435
Iowa	41,137	21,106	51.307
Kans.	30,589	15,611	51.035
Ky.	57,715	29,919	51.839
La.	42,270	21,596	51.091
Maine	15,723	8,022	51.021
Md.	27,236	13,928	51.138
Mass.	63,001	32,398	51.425
Mich.	87,446	45,034	51.499
Minn.	45,962	23,563	51.266
Miss.	48,320	24,514	50.733
Mo.	57,299	29,404	51.317
Mont.	10,029	5,103	50.882
Nebr.	23,327	11,947	51.215
Nev.	1,423	733	51.511
N. H.	7,768	3,943	50.760
N. J.	54,514	28,100	51.554
N. M.	13,190	6,692	50.735
N. Y.	184,344	94,620	51.328
No. Car.	78,753	40,354	51.241
No. Dak.	13,655	7,018	51.395
Ohio	101,103	51,770	51.205
Okla.	43,691	22,343	51.139
Oregon	13,179	6,825	51.787
Penn.	161,166	82,698	51.312
R. I.	10,215	5,246	51.356

TABLE 2 *(Continued)*

STATES	NO. OF BIRTHS	NO. OF ♂	PERCENT ♂
S. Car.	40,598	20,837	51.325
S. Dak.	12,850	6,604	51.393
Tenn.	53,314	27,400	51.394
Texas	114,721	58,836	51.286
Utah	12,695	6,509	51.272
Vt.	6,591	3,360	50.979
Va.	51,487	26,275	51.032
Wash.	22,396	11,591	51.755
W. Va.	41,774	21,395	51.216
Wis.	52,562	27,113	51.583
Wyo.	4,362	2,231	51.146
Total	2,155,105	1,105,489	
Mean	43,981.7	22,561.0	51.296

$$\sigma^2_o = \frac{\Sigma fd^2}{n-1} = \frac{.0003167473}{48} = .00000660$$

$$\sigma^2_T = \frac{.51296 \times .48704}{43{,}981.73} = \frac{.2498320384}{43{,}981.73} = .00000568$$

$$F = \frac{\sigma^2_o}{\sigma^2_T} = \frac{.00000660}{.00000568} = 1.12 = \text{Probability greater than } .05.$$

for the United States population was not altered in the period following the World War". It is true that Ciocco found a significant difference between the sex ratios of Negro and white populations and between those of certain other racial groups; but the question of genetic difference is hardly being tested in the above analysis, because it seems quite probable that the genetic composition of the Chicago population did not change strikingly from year to year during the interval considered.

In Table 2 are given the live birth sex ratios for each of the 48 states and for the District of Columbia for the year 1935. (There was no particular reason for choosing the year 1935 except that the data for that year are complete and were conveniently available.) Calculating the observed variance of these ratios and comparing it with the variance expected due to random fluctuations, we find that the former is 1.116 times greater than the latter; this deviation, however, is not a significant one. Thus even under the wide variety of environmental conditions

and genetic differences which obtain for the different states, the factors responsible for the deviations in live birth sex ratio do not vary, considering a period of one year, more than might be accounted for in terms of chance variations. Ciocco (1938) did not even find a significant difference between the mean sex ratio for each of 33 different states for a period of ten years and the mean for all the states for the same period of time. He concluded that "the sex ratio is not significantly and regularly influenced by the geographic, climatic, or other demographic differences to be found between the states in this country". The results of the above analysis are in general accord with this conclusion.

TABLE 3

Live births for U. S. Birth Registration Area for 1935

(From Births, Stillbirth and Infant Mortality Statistics 1935, U. S. Department of Commerce, Bureau of Census.)

MONTH	BIRTHS	NO. OF ♂	PERCENT ♂
Jan.	186,051	95,732	51.455
Feb.	171,058	87,400	51.094
Mar.	185,375	95,379	51.452
April	172,177	87,857	51.027
May	174,705	89,651	51.316
June	178,786	91,763	51.326
July	191,888	98,781	51.478
Aug.	192,449	98,943	51.413
Sept.	185,218	94,940	51.259
Oct.	177,945	91,163	51.231
Nov.	167,643	86,133	51.379
Dec.	171,810	87,747	51.072
Total	2,155,105	1,105,489	
Mean	179,592.1	92,124.1	51.296

$$\sigma^2_o = \frac{\Sigma fd^2}{n-1} = \frac{.0000274454}{11} = .00000250$$

$$\sigma^2_T = \frac{.51296 \times .48704}{179{,}592.1} = .00000139$$

$$F = \frac{\sigma^2_o}{\sigma^2_T} = \frac{.00000250}{.00000139} = 1.79 = \text{Probability greater than } .05.$$

Table 3 gives the percentages of male live births in the U. S. Birth Registration Area for each of the twelve months of the year 1935. These data include the births for all states and the District of Columbia. The variance of these percentages is only 1.794 times greater than that expected due to random fluctuations. This is not a significant deviation. In other words the factors responsible for the deviations of the live birth sex ratio of the United States are not affected sufficiently during one year by differences in the environmental conditions of the seasons to

TABLE 4

Live births for U. S. Birth Registration Area from 1923-1937

(From Statistical Abstracts of the United States, 1939. United States Department of Commerce, Bureau of Census.)

YEAR	NO. OF BIRTHS	NO. OF ♂	PERCENT ♂
1923	1,792,646	921,020	51.378
1924	1,930,614	992,431	51.405
1925	1,878,880	966,973	51.465
1926	1,856,068	953,638	51.379
1927	2,137,836	1,099,287	51.421
1928	2,233,149	1,147,625	51.390
1929	2,169,920	1,114,814	51.376
1930	2,203,958	1,131,976	51.361
1931	2,112,760	1,084,404	51.326
1932	2,074,042	1,063,885	51.295
1933	2,081,232	1,068,871	51.358
1934	2,167,636	1,112,703	51.333
1935	2,155,105	1,105,489	51.296
1936	2,144,790	1,099,465	51.262
1937	2,203,337	1,130,641	51.315
Total	31,141,973	15,993,222	
Mean	2,076,131.5	1,066,214.8	51.356

$$\sigma^2_0 = \frac{\Sigma fd^2}{n-1} = \frac{.0000040372}{14} = .000000288$$

$$\sigma^2_T = \frac{.51356 \times .48644}{2{,}076{,}131.5} = \frac{.2498161264}{2{,}076{,}131.5} = .000000120$$

$$F = \frac{\sigma^2_0}{\sigma^2_T} = \frac{.000000288}{.000000120} = 2.40 = \text{Probability less than .05.}$$

give a variance which is greater than that expected due to chance. These results agree in general with the findings of Lewis and Lewis (1906) who examined human sex ratios from different sources for seasonal variations and found no evidence of a consistent seasonal trend. They are also in agreement with the results of Ciocco (1938) who found by comparing the mean monthly sex ratios for two five-year periods that the monthly oscillations of the United States birth ratio "fall well within the expected deviations from the sex ratio of the total".

Having found no significant variation of the sex ratio for a local area over a period of twenty years or for the different states involving a period of one year or for the different seasons of a one-year period, it seemed of interest to test the variability of the sex ratios of the U. S. population as a whole over an interval of several years. In Table 4 are given the live birth sex ratios for the U. S. Birth Registration Area from 1923 to 1937 inclusive. (Only since 1932 are all states included in the U. S. Birth Registration Area.) The variance of the sex ratios for this area and for the period indicated is 2.397 times greater than that expected due to random fluctuations. This value of F has a probability considerably less than .05 and may therefore be considered significant. In other words it appears that the factors responsible for the deviations in live birth sex ratios from a 50:50 ratio do vary more than might be expected due to chance when the population considered extends over varied environmental conditions and over varied genetic sub-populations, and when the interval involved extends over a relatively long period of time. Ciocco (1938) did not find a significant difference between the mean U. S. sex ratio for different periods of a twenty-year interval and the mean for the whole interval but he did obtain evidence of a trend toward a decrease in the U. S. sex ratio from 1931 to 1934. As is evident from Table 4 this decline continued up to 1936. Whether this decline is the sole cause of the observed variability or whether it would have existed even so is not ascertained.

In the present analysis no attempt was made to determine what the factors are which are responsible for a deviation of the human sex ratio from a 50:50 ratio or to determine which of the various factors varies the most or the least under different environmental and genetic conditions. An attempt was made to determine the degree to which all the factors vary as a unit under different environmental and genetic conditions and during different intervals of time.

SUMMARY AND CONCLUSIONS

The variances (1) of the yearly live birth sex ratios for the Chicago Area from 1919 to 1938 inclusive, (2) of the live birth sex ratios of the 48 states and the District of Columbia for the year 1935, (3) of the monthly live birth sex ratios of the U. S. population as a whole for the year 1935, and (4) of the yearly live birth sex ratios of the U. S. population as a whole from 1923 to 1937 inclusive, were determined and compared with the respective variances of the live birth sex ratio expected due to random fluctuations. The general conclusion drawn is that the factors which are responsible for the deviation of human live birth sex ratios from the expected 50:50 ratio are not significantly affected (1) by the usual changes which occur in the external environmental conditions and the genetic composition of a local population over a relatively long period of time, (2) by the external environmental and genetic differences which exist between the different states of the United States, considering an interval of one year, and (3) by the external environmental differences which obtain during the different months of one year in the United States. However, the conclusion is drawn that the factors responsible for the deviations of human live birth sex ratios from the expected 50:50 ratio may be significantly affected by the external environmental variation or by the genetic variations which exist in the population of a country such as the United States, when a long interval of time is allowed.

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LOGARITHMIC CORRELATION COEFFICIENTS AND REGRESSION EQUATIONS*

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IT has been previously shown (1) that many skew frequency distributions can be simplified and made symmetrical by means of logarithmic analysis. The present paper applies and amplifies the findings of the previous study to skew correlation tables.

To simplify the presentation of the methods, specific examples are used. Streeter's data on the height and weight of human fetuses (2) and Pearl and Surface's data on the weight, length and breadth of eggs (3) were found suitable for a comparison of the arithmetic and logarithmic correlation methods.

Arithmetic correlation. The data of Streeter on the relationship of height and weight of 668 fetuses is shown in Table 1 by means of the standard arithmetic correlation table.

Casual inspection suffices to show that the correlation table is markedly skew. To prove mathematically that the correlation is skew, it is necessary to calculate the quantities r, η and ζ. The correlation coefficient, r, is 0.8432 while the correlation ratio, η, is much higher, 0.9808. The difference of the squares of these quantities or ζ equals 0.2510. The probable error of ζ as determined by Blakeman's formula (4) is 0.0631, which is 8.5 per cent of ζ. A correlation table has to be considered skew or non-linear if ζ is more than three times its probable error; in other words, if the probable error is less than one-third of ζ or 33⅓ per cent. In the case of Table 1, the probable error is only 8.5 per cent and therefore this correlation table is definitely skew.

Logarithmic correlation. It was shown in a previous paper (1)

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* From the Department of Pathology, Veterans Administration, Hines, Illinois.

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TABLE 1

Arithmetic correlation table of the height and weight of 668 human fetuses (Streeter's data)

WEIGHT (*gm.*)	HEIGHT (*mm.*)													TOTAL
	30–	60–	90–	120–	150–	180–	210–	240–	270–	300–	330–	360–	390–	
0–	43	111	107	93	97	3	..	..	..	..	..	..	..	454
400–	..	..	..	..	10	73	30	..	..	..	..	..	..	113
800–	..	..	..	..	..	..	14	13	..	..	..	..	..	27
1200–	..	..	..	..	..	..	..	8	4	1	..	..	..	13
1600–	..	..	..	..	..	..	..	1	11	5	..	..	..	17
2000–	..	..	..	..	..	..	..	..	3	8	..	..	..	11
2400–	..	..	..	..	..	..	..	..	..	6	10	..	..	16
2800–	..	..	..	..	..	..	..	..	..	1	4	1	..	6
3200–	..	..	..	..	..	..	..	..	..	..	4	3	..	7
3600–	..	..	..	..	..	..	..	..	..	..	1	1	..	2
4000–	..	..	..	..	..	..	..	..	..	..	..	2	..	2
4400–	..	..	..	..	..	..	..	..	..	..	..	..	..	0
Total	43	111	107	93	107	76	44	22	18	21	19	7	0	668

that the first step in the logarithmic analysis of a skew distribution is the construction of a logarithmic frequency table. Similarly, it is possible to construct a logarithmic correlation table (Table 2) of the data represented in Table 1. First, suitable logarithmic classes are determined (first row and column in Table 2). Then the arithmetic limits of these classes are found by determining the antilogarithms (second row and column). Finally, the number of cases that fall within each class can be readily obtained from the original data. The logarithmic correlation table is as easy to construct as the corresponding arithmetic table.

A comparison of the logarithmic Table 2 and the arithmetic Table 1 is interesting. The logarithmic table appears linear whereas the arithmetic one is definitely skew. Furthermore, it is observed that the logarithmic frequency distributions of height and weight appear symmetrical in Table 2 whereas the corresponding arithmetic frequency distributions are markedly asymmetrical. It would appear then that

TABLE 2

Logarithmic correlation table of the height and weight of 668 human fetuses (Streeter's data)

LOGARITHM OF HEIGHT		1.5–	1.6–	1.7–	1.8–	1.9–	2.0–	2.1–	2.2–	2.3–	2.4–	2.5–	2.6–	TOTAL
Height in mm.		31.62–	39.81–	50.12–	63.10–	79.43–	100.00–	125.90–	158.49–	199.53–	251.19–	316.23–	398.11–	
LOGARITHM OF WEIGHT	Weight in grams													
.6–	3.98–	1	10	2	..	..	..	..	..	..	..	..	..	13
.9–	7.94–	..	7	40	5	..	..	..	..	..	..	..	..	52
1.2–	15.85–	..	..	6	41	2	..	..	..	..	..	..	..	49
1.5–	31.62–	..	..	..	7	57	2	..	..	..	..	..	..	66
1.8–	63.10–	..	..	..	..	9	73	1	..	..	..	..	..	83
2.1–	125.90–	..	..	..	..	..	14	86	1	..	..	..	..	101
2.4–	251.19–	..	..	..	..	..	..	29	100	..	..	..	..	129
2.7–	501.19–	..	..	..	..	..	..	..	26	66	..	..	..	92
3.0–	1000.00–	..	..	..	..	..	..	..	..	5	32	2	..	39
3.3–	1995.3–	..	..	..	..	..	..	..	..	..	11	31	..	42
3.6–	3981.1–	..	..	..	..	..	..	..	..	..	..	2	..	2
3.9–	7943.3–	..	..	..	..	..	..	..	..	..	..	..	..	0
Total		1	17	48	53	68	89	116	127	71	43	35	0	668

Table 2 is a more satisfactory method of representing the data than Table 1.

The logarithmic correlation Table 2 can be analyzed mathematically by the same methods used for the arithmetic Table 1. The correlation coefficient r of Table 2 is found to be 0.9833. This is termed the logarithmic correlation coefficient. The logarithmic correlation ratio η is found to be approximately the same, 0.9843. ζ is then very low, 0.0021, with the probable error of 115.0 per cent. This high probable error shows that the logarithmic correlation table may be considered linear.

Logarithmic regression equations to correspond with the linear correlation Table 2 can be readily obtained by the standard methods. One equation is found to be

$$y = 2.999\,x - 4.1517 \qquad (1)$$

where y represents the logarithm of the weight (or log W) and x, the logarithm of the height (or log H). Equation (1) may therefore be written as

$$\log W = 2.999 \log H - 4.1517. \qquad (2)$$

This logarithmic regression formula can be changed to the geometric equation

$$W = 0.0000705\ H^{2.999}. \qquad (3)$$

Since the power of H is very close to 3, it would seem that the weight of the fetus is a linear function of the cube of the height. This conclusion is in all probability theoretically sound.

In this case then the use of logarithmic correlation leads to an equation which is simple to derive, has only two constants, and appears to have theoretical significance.

Multiple logarithmic regression equation. Another comparison of the methods of logarithmic and arithmetic correlation analysis is afforded by the data of Pearl and Surface (3). These investigators obtained the length, breadth and weight of 450 eggs and analyzed the data thoroughly using the standard correlation methods.

Pearl and Surface considered the arithmetic correlation table for weight and length to be non-linear with $r = 0.5797$, $\eta = 0.6127$ and $\zeta = 0.0393 \pm 33.8$ per cent (Table 3). The logarithmic correlation of the same data is, in contrast, definitely linear with $r = 0.5687$, $\eta = 0.5839$ and $\zeta = 0.0174 \pm 47.5$ per cent.

TABLE 3

The arithmetic and logarithmic correlation constants for the relationships of weight and length, weight and breadth, and length and breadth of eggs (Pearl and Surface's data)

	CORRELATION COEFFICIENT r	CORRELATION RATIO η	TEST OF LINEARITY $\zeta = \eta^2 - r^2$	PROBABLE ERROR OF ζ IN PERCENTAGE OF ζ
Weight-Length				
Arithmetic correlation	.5797	.6127	.0393	33.8
Logarithmic correlation	.5687	.5839	.0174	47.5
Weight-Breadth				
Arithmetic correlation	.8357	.8377	.0034	108.8
Logarithmic correlation	.8100	.8150	.0083	69.8
Length-Breadth				
Arithmetic correlation	.0837	....	....	....
Logarithmic correlation	.0865	....	....	....

The arithmetic correlation of weight and breadth was found by Pearl and Surface to be linear with $r = 0.8357$, $\eta = 0.8377$ and $\zeta = 0.0034 \pm 108.8$ per cent. The logarithmic correlation of the same data is also linear; $r = 0.8100$, $\eta = 0.8150$ and $\zeta = 0.0083 \pm 69.8$ per cent. It would appear that the data on length and breadth is not sufficient to determine which type of correlation is preferable.

The arithmetic correlation coefficient of length and breadth is, according to Pearl and Surface, small ($r = 0.0837$) and has a high probable error (37.8 per cent). Similarly, the logarithmic correlation coefficient is small (0.0865) with the high probable error of 36.5 per cent. There is then no appreciable correlation between the length and breadth of eggs.

Pearl and Surface studied the relationship of weight, length and breadth by multiple regression equations. Since the arithmetic correlation between weight and length is skew, the linear multiple regression equation was found unsatisfactory. Extensive calculations finally lead to the empirical parabolic regression formula

$$W = 1.0513\,L - 0.0426\,B - 0.00059\,L^2 + 0.0322\,B^2 - 56.6665 \quad (4)$$

where W = weight, L = length and B = breadth. It is to be observed

that this equation is empirical, has no theoretical significance, is difficult to derive and has 5 constants.

It has already been shown that the logarithmic correlations between weight, length and breadth are linear, and therefore one is justified in using the linear multiple logarithmic regression formula

$$\log \mathrm{W} = a + b \log \mathrm{B} + c \log L. \qquad (5)$$

The constants a, b and c were calculated by the standard methods as described by Pearl and Surface. The logarithmic formula was thus found to be

$$\log W = -3.3711 + 2.004 \log B + 1.067 \log L \qquad (6)$$

which can be converted to the geometric equation

$$W = 0.0004255\ B^{2.004}\ L^{1.067}. \qquad (7)$$

To determine whether this equation has any theoretical significance, it is first necessary to derive a theoretical formula for weight. According to Pearl and Surface, the egg may be considered a perfect prolate spheriod. The volume of the egg would then be

$$V = \frac{4}{3}\ \pi a^2 b \qquad (8)$$

where V is volume, and a and b are the semiaxis, a being the shorter. Formula (8) reduces to

$$V = 0.0005236\ B^2 L \qquad (9)$$

where V is expressed in cubic centimeters and the breadth and length in millimeters. Since the mean specific gravity of the egg was found by Pearl and Surface to be 1.0898, then

$$W = 1.0898\ V. \qquad (10)$$

Substituting for V in formula (9),

$$W = 0.0005706\ B^2 L. \qquad (11)$$

This is the theoretical equation for weight in terms of breadth and length.

It is to be observed that the form of the theoretical equation (11) is the same as that of the equation (7) which was derived by assuming logarithmic correlation among the component elements, weight, length and breadth. The exponents of B and L in the calculated formula (2.004 and 1.067 in formula 7) are approximately the same as those of the theoretical formula (2 and 1 in formula 11). It may be concluded that the geometric multiple regression formula (7) is a fairly good approximation of the theoretical equation (11).

TABLE 4

A comparison of the accuracy of the geometric formula and Pearl and Surface's parabolic formula in the calculation of the weight of 20 eggs

OBSERVED WEIGHT	DIFFERENCE BETWEEN OBSERVED WEIGHT AND THE WEIGHT CALCULATED BY		PERCENTAGE ERROR OF CALCULATED WEIGHT USING	
	Pearl and Surface's parabolic formula*	geometric formula**	Pearl and Surface's parabolic formula	geometric formula
56.88	+1.83	+2.79	3.22	4.91
52.96	— .37	+ .08	.70	.15
57.13	—1.74	— .99	3.05	1.73
51.07	— .37	+ .28	.72	.55
53.15	+1.28	+1.91	2.41	3.59
56.74	—1.03	— .41	2.82	.72
59.88	— .08	+ .89	.13	1.49
69.47	—1.11	+1.05	1.60	1.51
61.70	—1.49	— .43	2.41	.70
65.80	—1.12	+ .17	1.70	.26
69.51	—2.12	— .58	3.05	.83
59.83	—1.54	— .60	2.57	1.00
50.11	— .54	+ .11	1.08	.22
52.81	+ .11	+ .57	.21	1.08
52.31	+ .92	+1.60	1.76	3.06
49.63	— .14	+ .49	.28	.99
54.84	+ .26	+1.00	.47	1.82
54.90	+ .41	+ .06	.75	.11
54.08	— .14	+ .46	.26	.85
43.66	+1.48	+2.15	3.39	4.92
Total	18.08	16.62	32.58	30.49
Average	.90	.83	1.68	1.52

* $W = 1.0513\,L - 0.0426\,B - 0.00059\,L^2 + 0.0322\,B^2 - 56.6665$

** $W = 0.0004255\,B^{2.004}\,L^{1.007}$

Pearl and Surface tested the accuracy of their multiple parabolic regression formula (4) by calculating the weights of 20 eggs chosen at random. The differences between the calculated and the observed weights are shown in Table 4. The average error in the prediction of the weight is 0.90 grams or 1.68 per cent of the actual weight.

The multiple geometric regression formula (7) was similarly tested by calculating the weights of the same 20 eggs. The differences between the observed and the calculated weights were determined and are shown in Table 4. It is seen that the average error is 0.83 grams. The average percentage error is 1.52. It would appear from this test that the geometric formula (7) is as satisfactory in predicting the weights of eggs as Pearl and Surface's formula.

It has been seen that the parabolic formula of Pearl and Surface has 5 constants, is empirical and is laborious to calculate. The geometric formula, in contrast, has only 3 constants, has definite theoretical significance and is as easy to calculate as any linear regression equation. Both formulae are equally accurate. It seems then that the logarithmic correlation methods and the derived geometric formula are preferable in this case.

Multiple logarithmic correlation coefficient. The multiple geometric regression formula (7) is useful in summarizing a large number of observations. To determine the reliability of this equation, it is necessary to calculate the multiple logarithmic correlation coefficient R. Using the logarithmic correlation coefficient of Table 3, it is found that the multiple logarithmic coefficient for the weight of eggs is 0.9506. The high value is consistent with the previous finding that the multiple geometric regression formula permitted fairly accurate predictions of the weight of eggs. This high regression coefficient would be expected since the length and breadth are the two most important factors which determine the weight of the egg. If a third factor, specific gravity, had been taken into consideration, the regression coefficient would have been even higher.

SUMMARY AND CONCLUSIONS

An arithmetic correlation table that is linear can be readily analyzed and represented by the correlation coefficient and simple regression equations.

Many arithmetic correlation tables in biology are, however, skew and are more difficult to analyze mathematically. Regression equations for such tables are difficult to derive.

The skew arithmetic correlation tables can be often converted into a symmetric correlation table by using logarithmic instead of arithmetic classes. Such a symmetric logarithmic correlation table is analyzed mathematically by the same methods as a symmetric arithmetic correla-

tion table. The regression equation of a logarithmic table can be easily calculated and can be represented by the geometric formula $y = ax^b$. Furthermore, the regression equation of the logarithmic correlation table has, at least in some cases, theoretical significance.

By means of logarithmic correlation tables, it was found that the relationship between the weight (W) and height (H) of human fetuses may be represented by the formula $W = 0.0000705\ H^{2.999}$; that is, the weight varies as the cube of the height. The relationship of the weight (W), length (L) and breadth (B) of eggs was found to be:

$$W = 0.0004255\ B^{2.004}\ L^{1.067}$$

This formula is approximately the same as the theoretically derived equation:

$$W = 0.0005706\ B^2\ L$$

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RACIAL DIFFERENCE IN RESISTANCE TO RESPIRATORY INFECTION

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DIFFERENCES in racial or species immunity to disease are well known to the botanist and to the zoologist: indeed the seeking of hereditarily resistant strains is one of the chief techniques of the agronomist in overcoming plant diseases. In human biology it is much more difficult to conclusively demonstrate racial differences in immunity because of the extreme difficulty in obtaining controlled conditions. For example, it has been pointed out that tuberculosis is more prevalent among Negro than among white groups. However, it has also been proved that various secondary factors as low economic standards, inadequate housing and poor sanitation which are known to increase the susceptibility to tuberculosis infection play a relatively greater role in Negro than white communities. It is because of these contributory factors that observations concerning the resistance of white and Negro are ordinarily not comparable.

At the New York State Training School for Boys, Warwick, where Negro and white boys of the age of approximately 12 to 17 years live together under identical and controlled conditions, studies of two waves of respiratory infection among the boys proved to be significant for statistical and clinical comparison.

The boys of the training school are housed without racial segregation in fourteen cottages and one reception unit. In the cottages about two thirds of the boys occupy the dormitory and the others sleep in single rooms. No preference is given either race with respect to assigning single rooms. In the reception unit, all boys have single rooms. For seven hours a day a boy associates with boys of other

* The writer is deeply indebted to Dr. Richard L. Jenkins, Psychiatrist of the N.Y.S. Training School for Boys, for his advice and help in the statistical part of the paper.

cottages while in school or on work assignment. He spends in the company of the other boys of his own cottage approximately seventeen hours a day, except for occasional recreational activities in larger groups. Here again, with very few exceptions, racial groups are mixed.

Before they came to the training school most of the boys had lived in urban districts such as New York City and vicinity. None of the Negro boys who had respiratory infection had recently come from the south. Six boys, observed during the first wave of respiratory infection, were born in the south, one boy was born in Puerto Rico. Individuals of mixed white and Negro blood were included in the classification of Negro, but the majority of this group gave little evidence of white blood.

At the morning sick call, which is held on all work days and to which boys are referred from the cottages, the sick are recognized and taken to the hospital of the training school. Without exception, any boy with fever is admitted to this hospital.

After an interval of more than four weeks without a hospital admission for respiratory infection, on December 23rd, 1940, the first patient with this disease was admitted. The first wave of subsequent admissions increased until December 29th and descended to a trough on the 2nd day of January, 1941. The second wave of admissions started on January 3rd, reached its peak around January 9th and ceased on January 11th.

The census of the population of the school at the beginning of the first observation period was 338 boys, 207 (61.24 per cent) being Negroes and 131 (38.76 per cent) being white boys. On January 3rd, when the second series of admissions began, there were 408 boys in the institution, 240 (58.82 per cent) of them Negroes, 168 (41.18 per cent) white boys. Sixty-five boys (35 Negroes, 30 white) had left for a Christmas visit shortly before the first census had been taken. The second census includes the number of boys who returned from this visit as well as five other commitments to the school. Inasmuch as the Negro-white ratio did not suffer significant changes, the difference can be neglected.

During the first wave, of 27 cases of respiratory infection admitted to the hospital, 24 were Negroes. We have compared the proportion of Negro boys in the group afflicted with the proportion expected for the first observation period. The simplest assumption was that the Negro boys should be affected in the same proportion in which they are in the

training school. This would have given us the number of 16.54 Negro boys. However, the number of Negroes observed with the disease was 24. The Negro group contributed 61.24 per cent of the total population December 24th. The Negro group provided 88.89 per cent of the boys affected with the disease.

We must consider the fact that some cottages run higher than others in their proportion of Negro boys and that some cottages have several boys ill while others have none. This suggests the possibility that the foregoing discrepancy might be the spurious results of the concentration of infection in preponderately Negro cottages. This is taken into account in Table 1 which reports the percentage of Negroes in each cottage on December 24th and the number of boys from that cottage admitted to the hospital. If there were no racially selective factor in the infection, then the expected number of Negroes ill in each cottage would be the product of the percentage Negroes and the number

TABLE 1

Expected and observed number of sick Negroes from each cottage from December 23rd to January 2nd

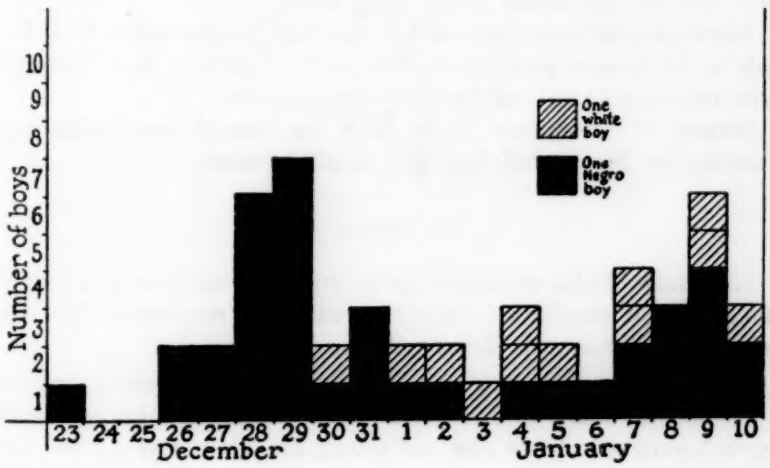
COTTAGES	PERCENT NEGRO		NUMBER SICK	EXPECTED NUMBER OF NEGROES	OBSERVED NUMBER OF NEGROES
A–1	0.56	×	1	0.56	1
A–2	0.50	×	1	0.50	1
A–3	0.48	×	4	1.92	3
A–4	0.83	×	1	0.83	1
B–1	0.52	×	2	1.04	2
B–3	0.29	×	2	0.58	1
B–4	0.34	×	2	0.68	2
C–1	0.50	×	0	0	0
C–2	0.71	×	4	2.84	4
C–3	0.89	×	0	0	0
C–4	0.88	×	2	1.76	2
D–1	0.91	×	0	0	0
D–2	0.85	×	5	4.25	4
D–4	0.82	×	3	2.46	3
Reception	0.64	×	0	0	0
Totals				17.42	24

ill. Totalling these products gives an expected value 17.42. We may consider the likelihood that a deviation from 17.42 would be as far as the observed value 24. The standard error of our expectation 17.42 out of 27 is 2.49. The difference between the expected value and the observed is more than three times this standard error and may be accepted as of statistical significance since the chance that such a discrepancy would occur on a random basis is distinctly less than 1 in 1000.

The proportion of Negroes and the incidence of infection in work groups were also examined, but no concentration of infection was discovered in work or school groups which might account for the overwhelming incidence of Negroes who became ill.

The second wave of admissions shows no significant difference in the racial distribution of the affected cases as Table 2 shows. During the first period there had been no admissions of white boys until the

TABLE 2



8th day. The second period starts with one admission, a white boy. From the total of 240 Negro boys, 14 (5.83 per cent), and of 168 white boys, 9 (5.35 per cent) were admitted during the second period. In correlation to the population of the cottages, based on the census of January 3rd, 13.39 Negro boys could be expected to get the infection. Fourteen cases were observed.

In addition the clinical picture impressed us with certain differences between the first and second series of infections. Though our observations are not available for statistical procedures, we should like to mention them as an additional means of distinction.

During the first period, the boys were found to have mainly an affection of the trachio-bronchial system. Three had an additional tonsilitis. When complaints of headache were given, the pain was described as dull and of general type without special reference to any particular region. The areas of the sinuses were not tender to pressure. During the second period, the majority of cases showed the symptoms usually met in mild forms of sinusitis. The boys would complain of definite frontal headache and watering eyes. Pressure over the frontal sinuses elicited pain. Application of dry heat to this area and medication of ephedrine nose drops usually brought relief.

Comparison of the fever curve of the two observation periods showed no significant differences. The lytic type was prevalent in both. The average duration of fever for the first group was 63.33 hours and for the second group 53.43 hours.

Bacteriological examinations for hemolytic streptococcus have been made on 15 random cases of the first period. In only three cases this bacterium could be isolated from the throat exudate.

Sixteen of twenty-four Negro boys, and one of three white boys, ill during the first period, had been tonsillectomized.

SUMMARY

1. Standard living conditions for Negro and white boys at a training school made it possible to study two waves of respiratory infections under almost laboratory like conditions.

2. During the first wave of infection, an overwhelming majority of the infected cases were Negroes. The rate of infection among the Negro boys was 11.6 per cent and among the white boys 2.3 per cent. This difference is too large to be accounted for on a basis of chance. Obviously it may be due to racial differences but early differences in environmental factors cannot be excluded. It would seem to indicate that the resistance against the particular infective agent responsible for the first epidemic is lower in the Negro race than in the white race.

3. The second wave of infection, which immediately followed, affected both races in almost equal proportions.

4. The clinical features of the two waves of respiratory infection showed certain differences. While the majority of cases of the first period presented symptoms of trachio-bronchial infection, the cases of the second period were mostly affected with frontal sinusitis.

NOTES

A NOTE ON THE ESTIMATION OF DYSPLASIA IN HUMAN PHYSIQUES: SHELDON'S METHOD AND THE ANALYSIS OF VARIANCE

IN HUMAN BIOLOGY, 13 (3): 405-410, 1941 ("A Note on Sheldon's Method for Estimating Dysplasia"), J. Zubin and M. Taback make two interesting theoretical points. First, Sheldon's method (1) is simplified from a long algebraic summation to the formula

$$D = 4\ (P'_1 - P'_5) + 2\ (P'_2 - P'_4) \qquad (1)$$

where D represents dysplasia for a component and $P'_1 \ldots P'_5$ the ratings for that component in order of magnitude. It is then shown that

$$D = 10\ AD' + 2\ R \qquad (2)$$

where AD' is the average deviation about the median rating, and R the range. While it is instructive to know the nature of Sheldon's measure of dysplasia in terms of the familiar statistics of equation (2), equation (1), Zubin and Taback's "simplified" formula, saves no time in computation. The algebraic formula for the original method gives a false impression that it is laborious to compute in actual practice.

Secondly and more importantly, the authors propose to substitute for this estimation the analysis of variance technique, using interregion variance as an index of dysplasia. Two advantages are claimed for this procedure: (1) the reliability of the measure of dysplasia can be determined by comparing the interregion with the intraregion variance; (2) the dysplasia of individuals and groups can be compared directly, estimating the significance of differences by the F ratio.

In respect to reliability, the authors admit that "where only one measure is available for each region, the reliability of the dysplasia measure cannot be determined." As possibilities for independent

ratings, they suggest different photographs of the same individual or different observers' ratings of the same photograph. The whole discussion is thus purely theoretical, since in practice (a) a standardized photographic technique is employed; and (b) the anthroposcopic ratings differ negligibly as between trained observers, inasmuch as the former are merely estimates of seventeen measurements, which in any case determine the final rating. Nevertheless, the theoretical implications are important. Assuming two independent ratings for the same individual, would comparison of interregion with intraregion variance indicate the reliability of the former as a measure of dysplasia? The authors admit in a footnote that "the intraregion variance would be better designated as between-rating variance . . . further . . . since the ratings are rather discrete and crude, it is too much to hope that the distribution of repeated ratings would satisfy the prerequisites of normality that the analysis of variance technique demands." Two further points not mentioned are that any two cameras or observers would differ *systematically,* and that certain regions (especially region I, the head and neck) are more variable internally than others. Both considerations enhance the unlikelihood of a normal distribution of ratings.

Sheldon suggests (p. 94) a measure of intraregion dysplasia which Zubin and Taback ignore; namely, the lowest mean rank of the somatotype of a given region. A rank of "zero" means that the indicated somatotype exactly fits the anthropometric criteria. The higher the rank, the more dysplastic is the region. On the unlikely assumption that this intraregion measure varies normally and homogeneously enough as between individuals, components, and regions to warrant use of the analysis of variance, the *interaction* variance would still indicate the reliability of interregion variance better than would the intraregion. Both inter- and intraregion variance would then be measures of dysplasia, and interaction variance could serve as the error term for either.

Direct comparison of individual and group variances by the F ratio test of significance assumes the normal distribution and homogeneity of variance of individuals, components, and regions in respect to dysplasia (Lindquist, (2)). These conditions are not in fact satisfied, contrary to the assertion of Zubin and Taback. The curve of total dysplasia in the male population is considerably skewed positively. Sheldon's d-index of dysplasia, not mentioned by Zubin and Taback, is more nearly normal; this is a division of the range of dysplasia into seven steps, numerically equal except at the extremes. However, total dysplasia

varies systematically in *individuals* according to sex (females are more dysplastic), somatotype (a 444, to mention only one, is almost always very dysplastic), and "constitutional" conditions (e.g., schizophrenes show more dysplasia than normal individuals). As for *components*, "the third component uniformly shows more dysplasia than the second, and the second shows more than the first" (Sheldon, p. 70). Moreover, the components vary independently only within certain limits, inasmuch as their sum must be between 9 and 12. As between *regions*, some (as already noted, region I) are intrinsically more variable than others; and the differences in dysplasia between sexes, somatotypes, and constitutional deviants occur in specific regions (e.g., there are "several recurrent and apparently characteristic dysplasias", by region and component, among schizophrenes).

Thus, both for reliability and comparison, Sheldon's method of computing dysplasia, involving no such assumptions as the analysis of variance, is probably as good an estimate as can be expected at the present stage of gathering data for the establishment of norms. Use of the roughly normally distributed d-index, for which estimates of reliability can be easily devised, is justified by the results already obtained. Nevertheless, as Zubin and Taback rightly point out, the more familiar and simpler analysis of variance technique is preferable. When distributions are established for each sex, somatotype, region, component, constitutional state, and the several combinations of these variables, many distributions will undoubtedly be normal. Thus, for example, the endomorphic dysplasias of normal and schizophrenic females in region V, or the total dysplasia of two normal male 444's might be compared by the analysis of variance and the F ratio; but only if the necessary conditions of normality of distribution and homogeneity of variance are satisfied.

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COMMENTS ON AN UNHAPPINESS OF MR. FRASER ROBERTS

MR. Fraser Roberts[1] has evidently been subjected to such a degree of mental distress by our temerity[2] in questioning established dogma on the intelligence-fertility correlation that he has neglected to determine in at least one important respect just what the offending article said. Thus it seems in the public interest to make a brief restatement of the logical structure of the problem dealt with, in the hope of preventing misunderstanding.

The objective was to take as nearly unselected a tested population as was available and determine whether there were significant differences in the number of offspring produced, in as long a period as was available, between its brighter half and its duller half. The answer is *No*. We labor under no delusions that this is the last or the best study in the field. However, to our knowledge it is the most nearly adequate one *on this topic* (as opposed to the topic of correlation between intelligence and sibling number) to date; and we infer from these facts that there is no objective evidence (however much faith there may be) *at the present time* for the proposition that the duller sections of the *population* (note that we are not saying "the parents of duller children") are reproducing faster than the brighter sections. It is entirely conceivable that when more adequate studies are made they will provide such evidence; but if it now exists we do not know of it, and would be grateful to Mr. Fraser Roberts for the reference.

Two of Mr. Fraser Roberts' specific disgruntlements need be only briefly noted: (1) The population studied is not large, but neither is it small; we were under the impression that determining the reliability of sampling by epithet had become obsolete, having been superseded by well-known statistical formulae. We know of no reason to call the composition of the population "peculiar": that a considerable proportion had no children we are inclined to look upon as a possible clue to the differences in reproductivity, rather than a damaging circumstance. We are, as mentioned before, concerned with segments of the *population*, not with that fraction of it which becomes parents; the reason why it is the population which is the object of our concern is that the practical measures which derive from studies on these matters are virtually

[1] HUMAN BIOLOGY, Vol. 13, p. 410, 1941.
[2] HUMAN BIOLOGY, Vol. 12, p. 114, 1940.

certain to be in terms of the population and not of fractions of it. (2) The population studied is undeniably selected, and we thought we presented a full evaluation of this circumstance. The observation may be repeated that while the boys are rather severely selected, the girls have a mean IQ only two points above the mean for the generality and the same variance. The American high school is possibly rather more nearly representative of the American people than Mr. Fraser Roberts realizes.

The third disgruntlement, involving our arithmetic (which is undoubtedly as subject to error as that of better men, although the material was carefully checked), appears to rest on Mr. Fraser Roberts' failure to comprehend what he was reading. Having split the population at the IQ median and enumerated the offspring of both halves, we did not thereupon compute the correlation as —.12 and declare it significant, thereby leading ourselves away from our original contention; on the contrary, we eschewed the use of the correlation technique in this situation. And our "dark suspicion" of the sibling coefficient could be less histrionically expressed as an attempt to learn whether absence of intelligence-offspring correlation can coexist with negative intelligence-sibling correlation; if it can, it becomes possible that any or all of the classical (indirect) studies, which unquestionably establish the latter, may have been drawn from populations in which the former was also the case. That is (the answer being affirmative), Mr. Fraser Roberts' admirable studies, demonstrating beyond doubt that brighter children come from smaller families, shed no light whatever on the more important demographic question of whether these brighter children can be expected to have more or fewer children than their duller schoolmates. The coefficient of —.12, which we believe to be correctly calculated and on the borders of significance, is that between intelligence and *sibling* number; Mr. Fraser Roberts has "disproved" it by showing that the correlation between intelligence and *offspring* number (which we did not calculate) is —.02. You can't convict a man of error in a problem he did by proving that the answer to one he didn't do was something else.

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RECENT LITERATURE

USEFUL IN THE STUDY OF HUMAN BIOLOGY

In this department will be noticed the books and reprints which are currently received at the Editorial Office. Publications intended for notice here should be addressed to Dr. Lowell J. Reed, Chairman of the Board of Associated Editors of Human Biology, 615 North Wolfe Street, Baltimore, Maryland, U. S. A.

I. EVOLUTION AND GENETICS

1. *Evolution — General Principles*

CATTELL, JAQUES [Editor]. Biological Symposia, Volume IV. I. Population problems in protozoa. II. Experimental control of development and differentiation. III. Theoretical and practical aspects of polyploidy in crop plants. IV. The species concept. With a Foreword by Sewall Wright. *Lancaster, Penna.* (Jaques Cattell Press), 1941. Pp. vii + 293. 9⅝ × 6¼ inches. $2.50. [Bibliographies at the end of some of the articles.]

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II. GROUP BIOLOGY

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